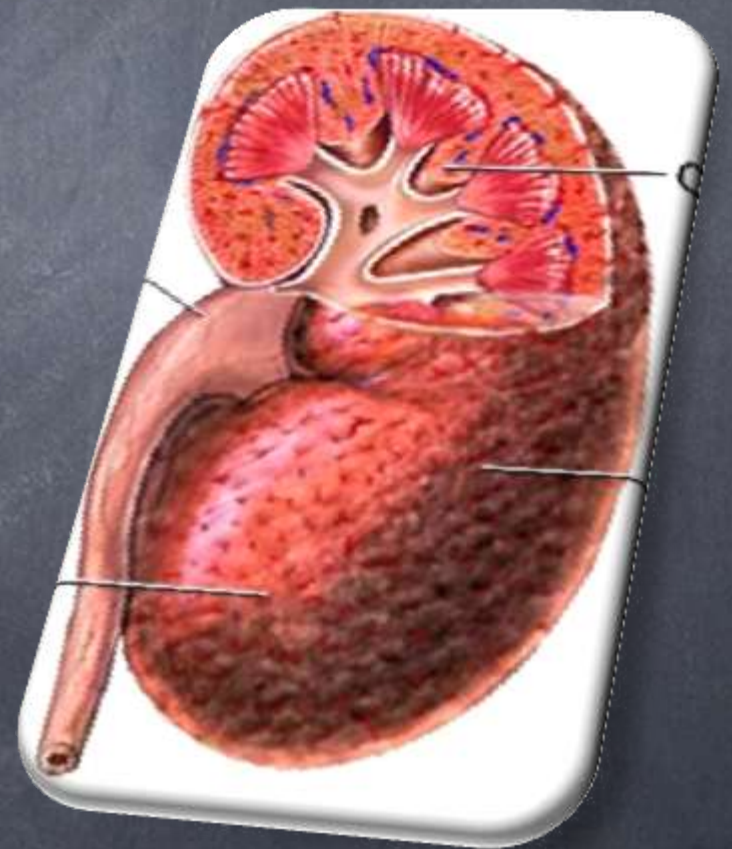


Pathology of Hypertensive Kidney Disease

Wesam Ismail, MD

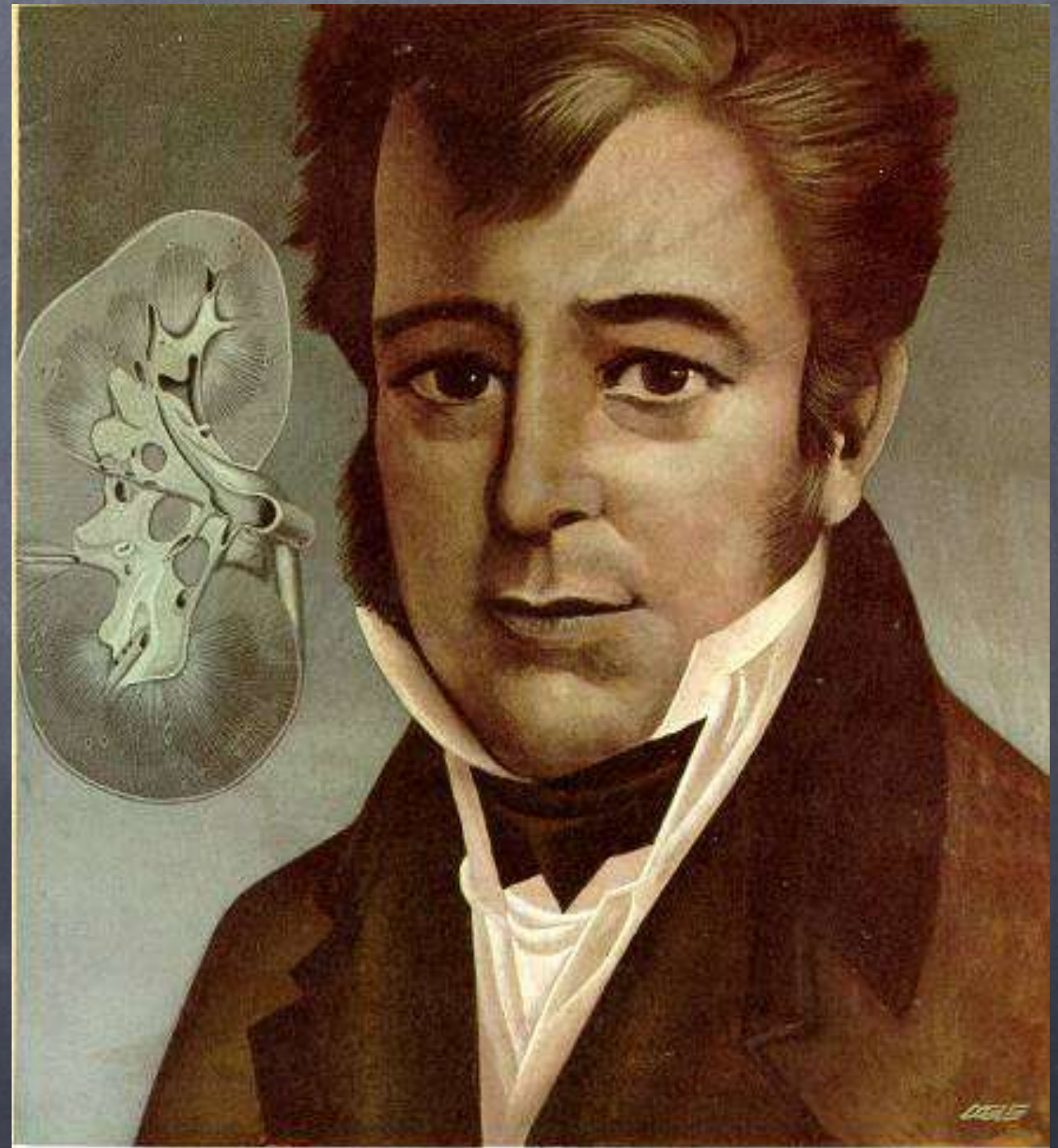
*Pathology Department
Bani Suef Univeristy*



The Kidney and Hypertension

Sir Richard Bright
(Guy`s Hospital Rep
1836;1:380):

„...renal dysfunction is
the primary cause of
hypertension“



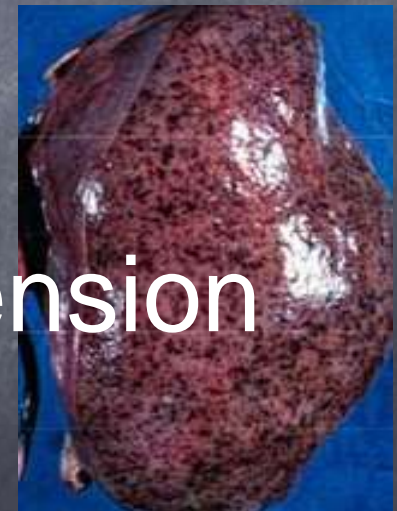
Hypertension and the Kidney

Essential Hypertension



Hypertensive Nephropathy “*Benign Nephrosclerosis*”

Accelerated Phase “*Malignant*” Hypertension



**Secondary
Hypertension**

Hypertensive Nephropathy

“Benign Nephrosclerosis”

- ❑ The kidneys in benign nephrosclerosis are typically reduced in size between (120 and 250 g)
- ❑ The two kidneys are usually affected equally
- ❑ The capsular surface is most commonly finely granular “leather like” reflecting disease in small arteries and arterioles

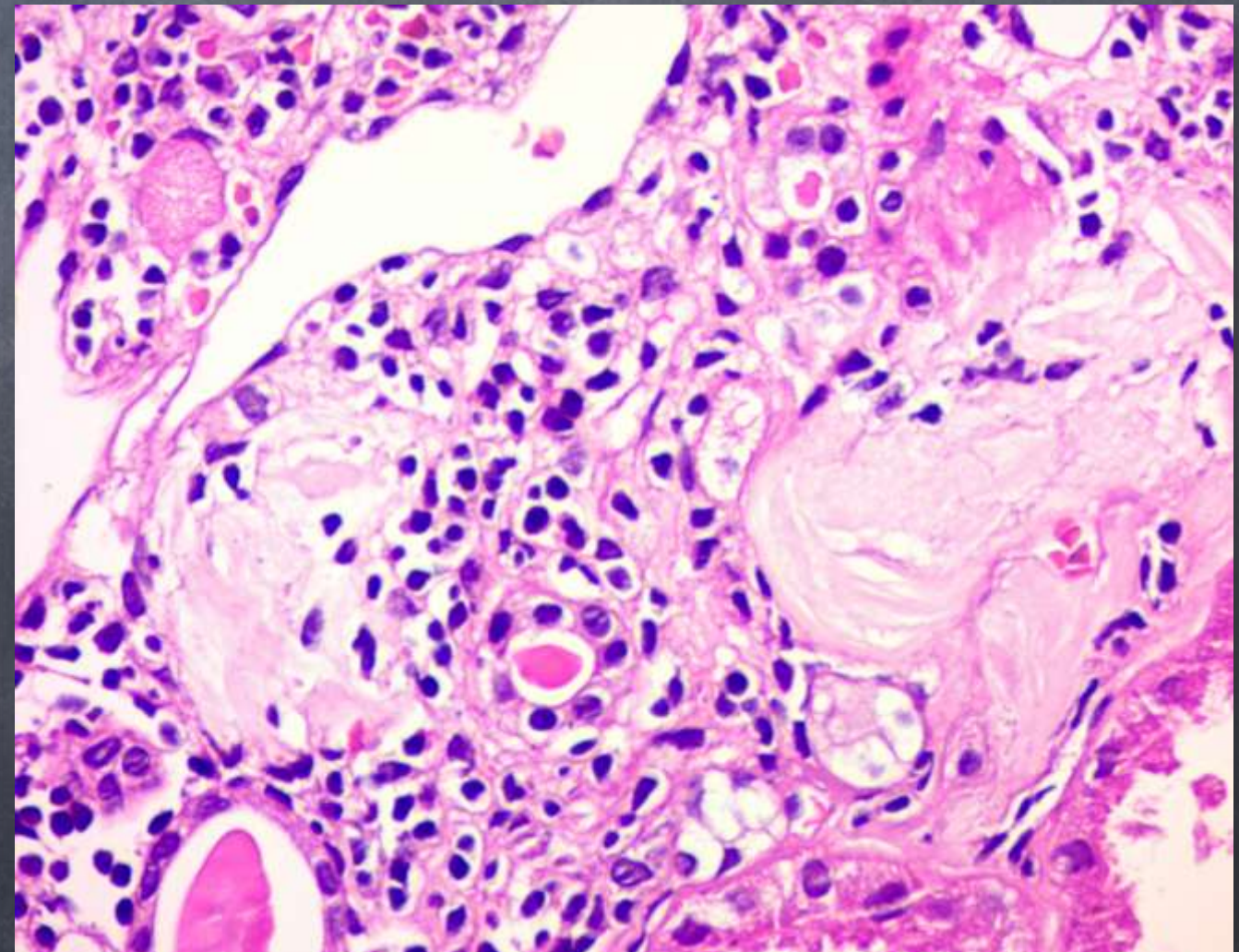
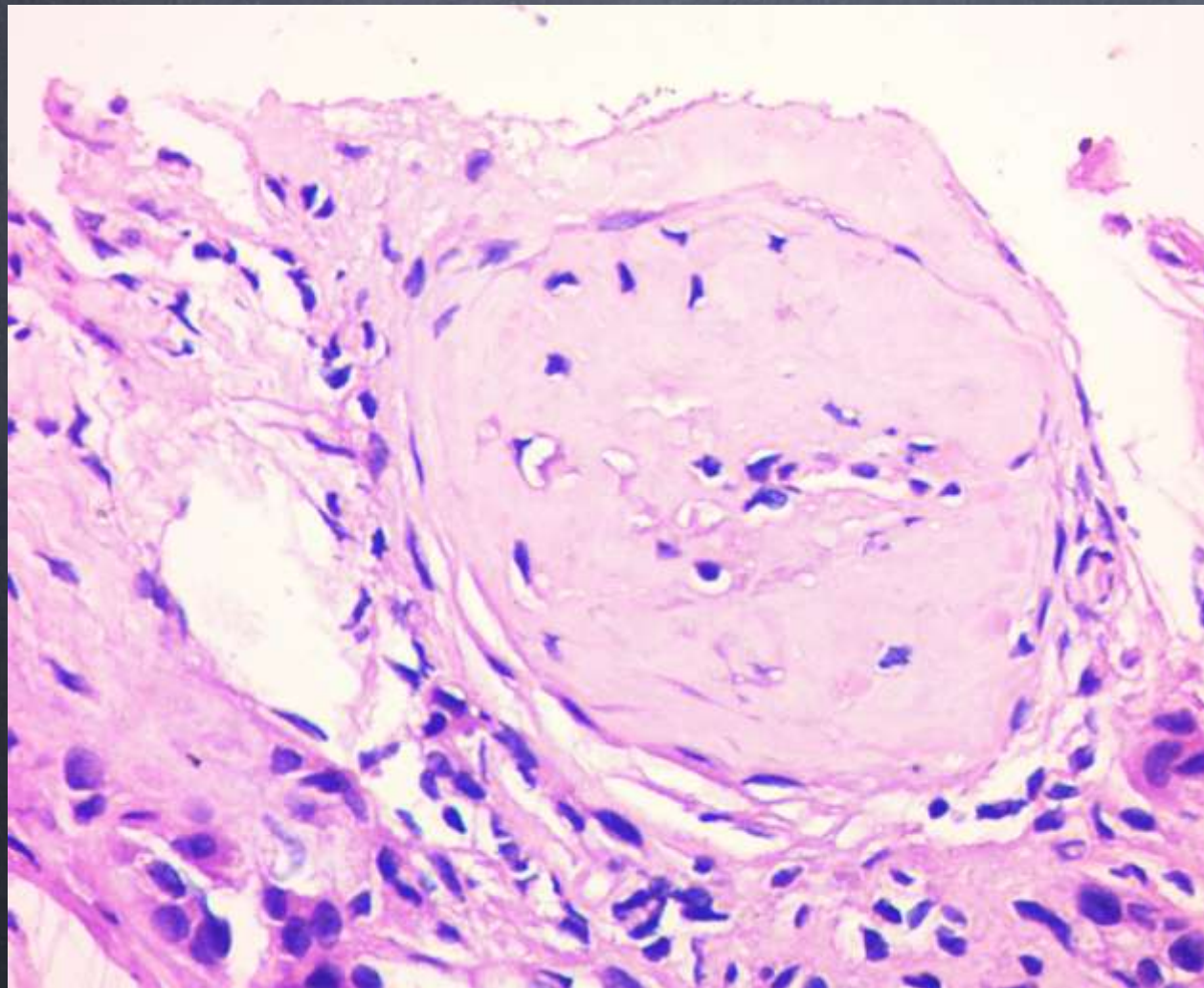
zones of relatively preserved or even hypertrophied renal parenchyma alternating with neighboring nephrons with diseased arterioles, which are scarred and depressed



Hypertensive Nephropathy

“Benign Nephrosclerosis”

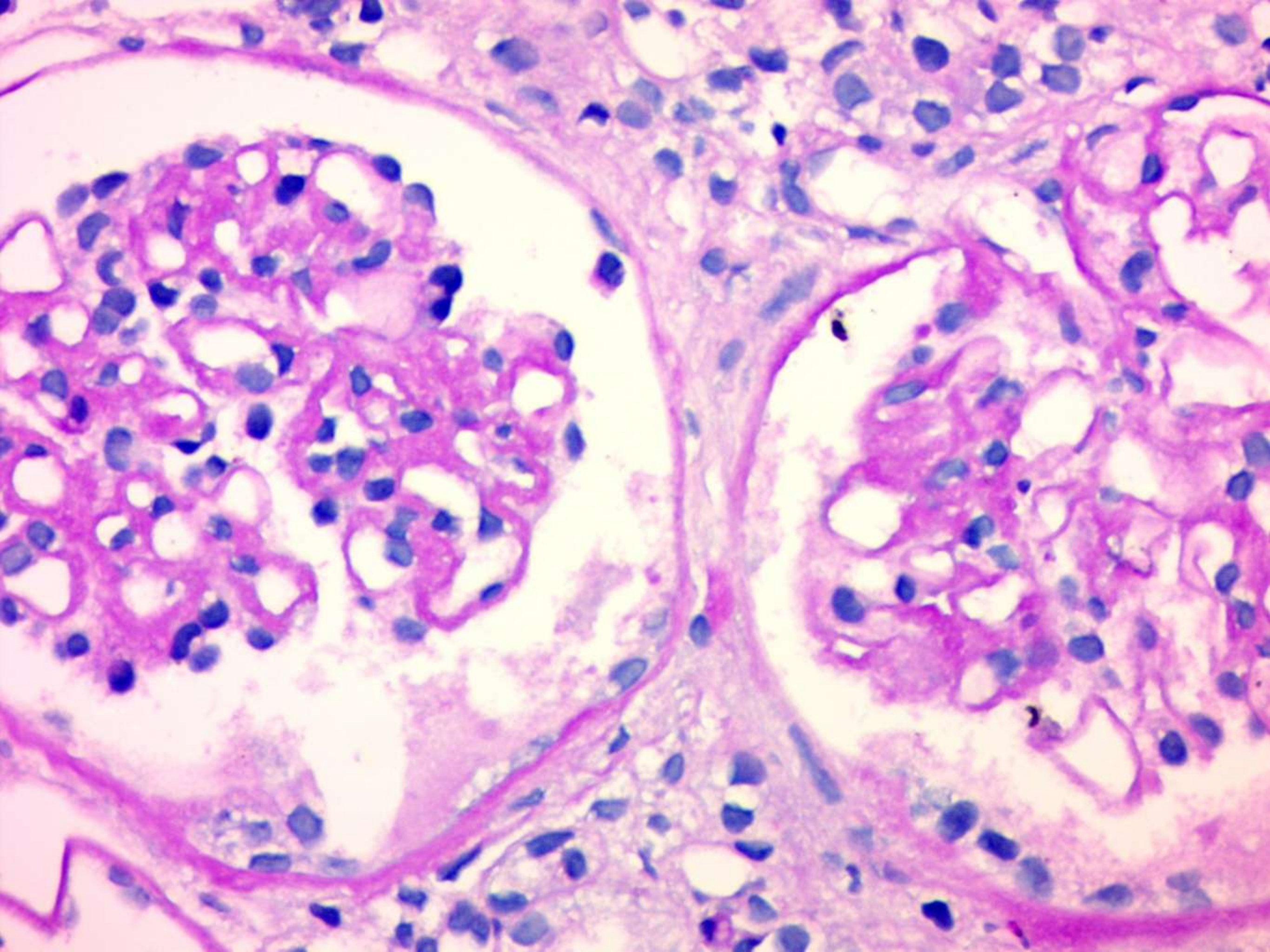
- ❑ The glomeruli may be normal or may simply show age-related changes (few sclerosed glomeruli)
- ❑ Sclerosed glomeruli may disappear “*disappearing glomeruli*” that appear to merge into the surrounding renal parenchyma

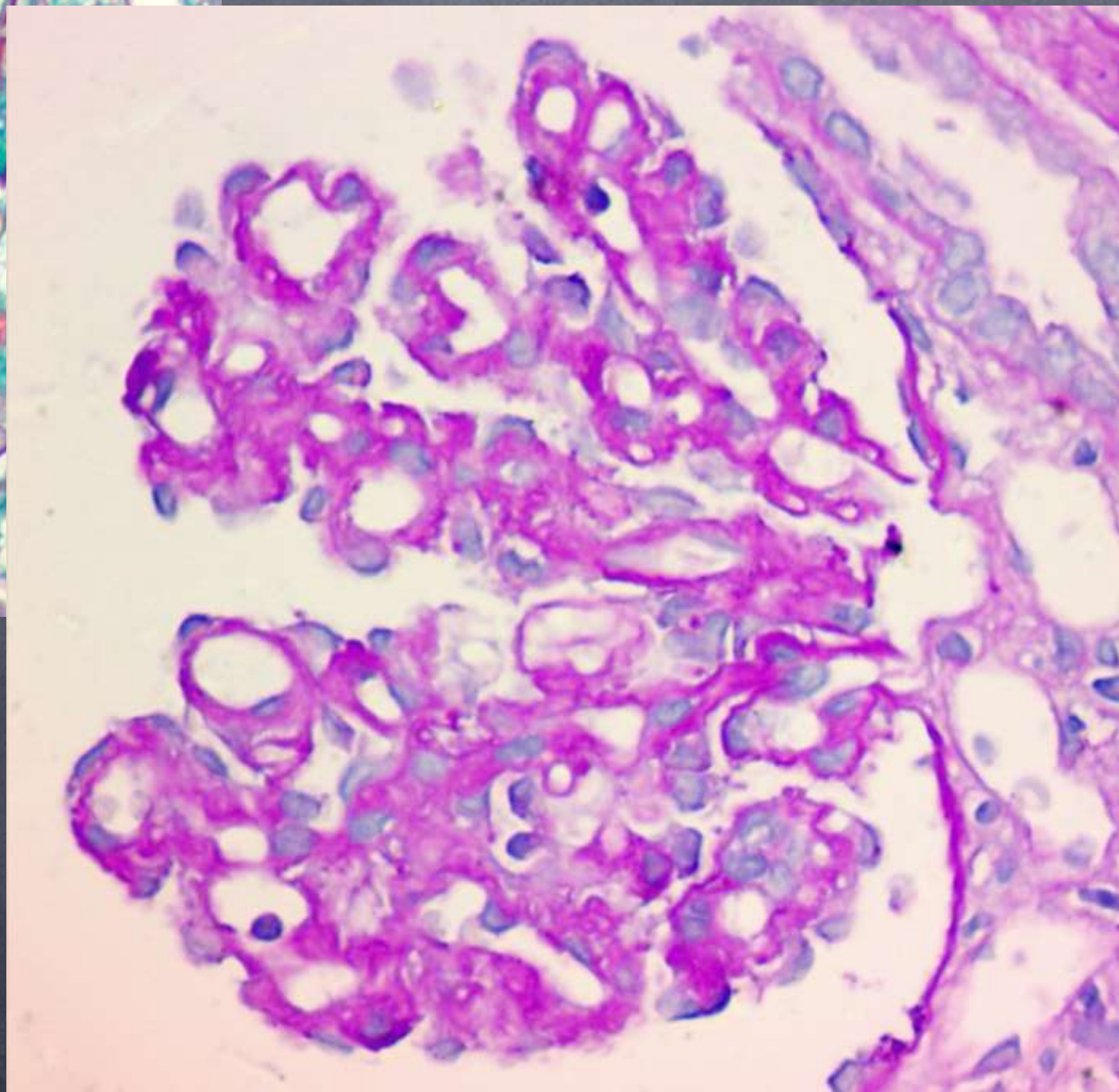
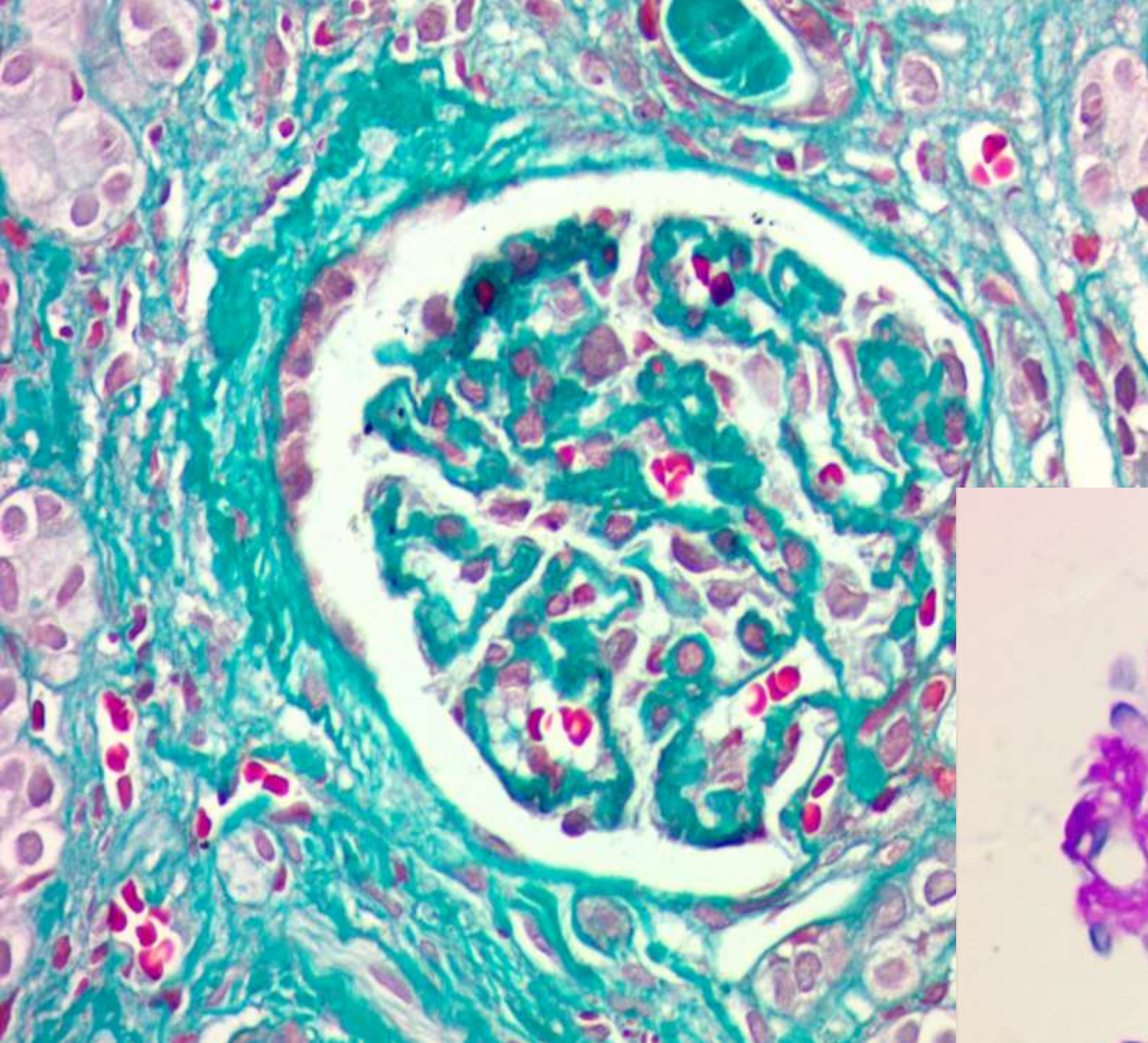


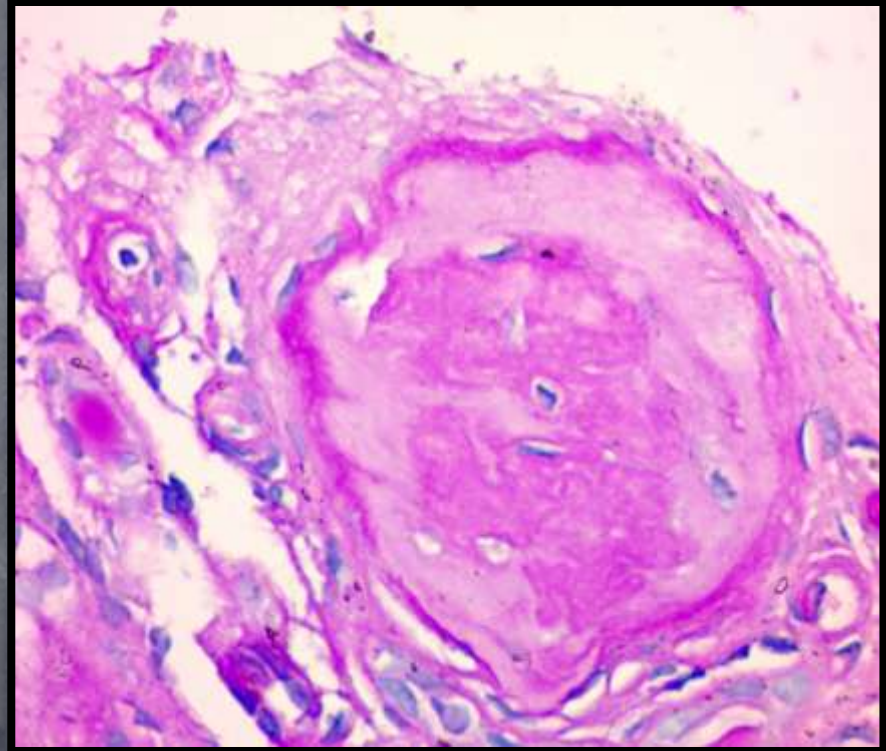
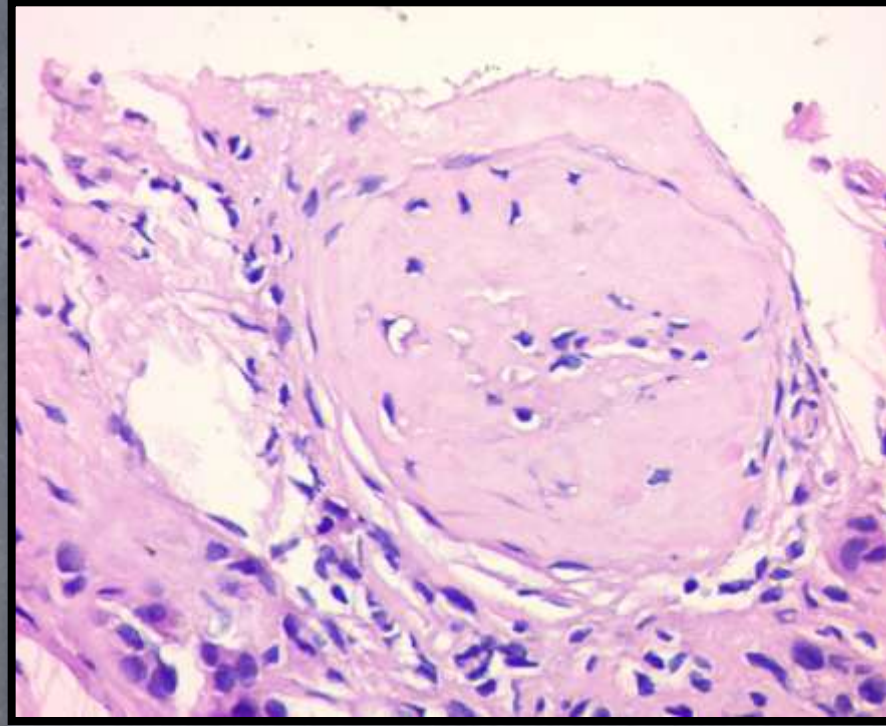
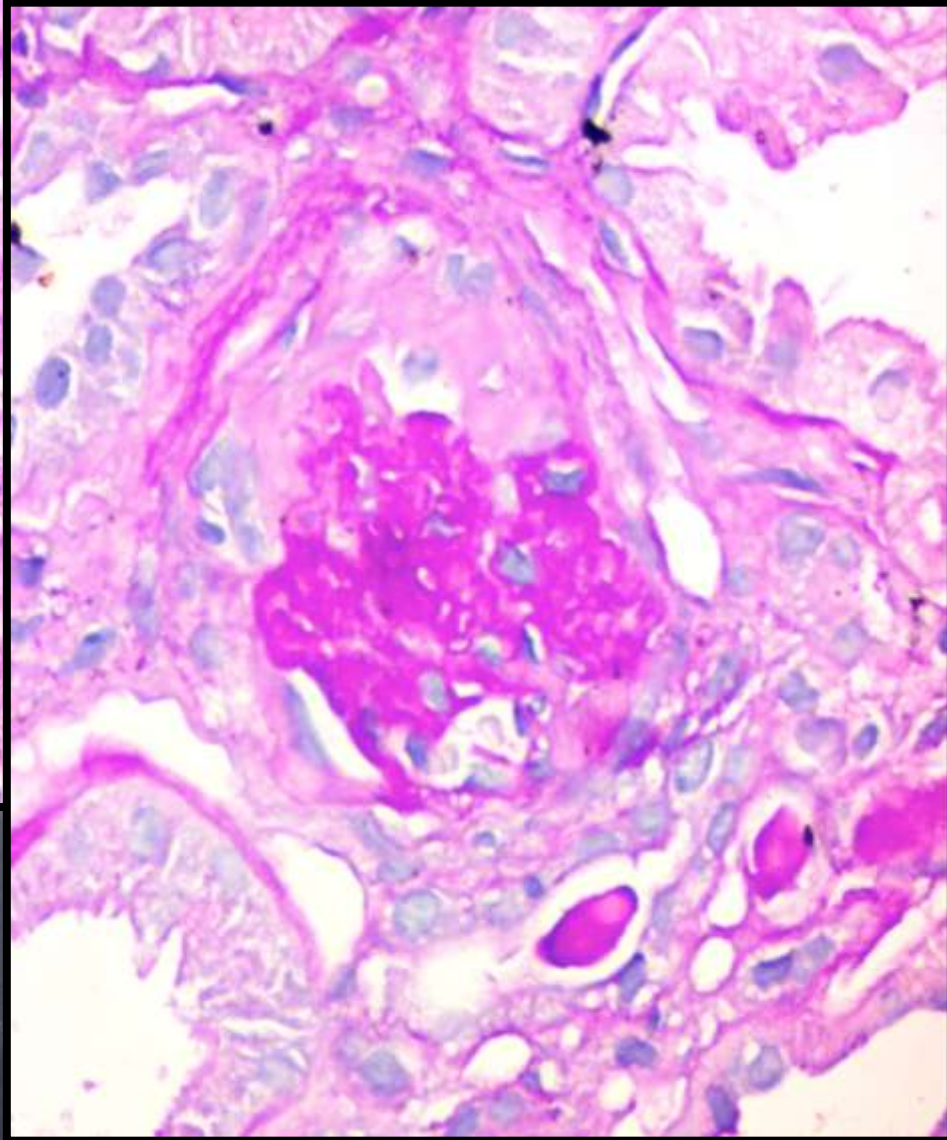
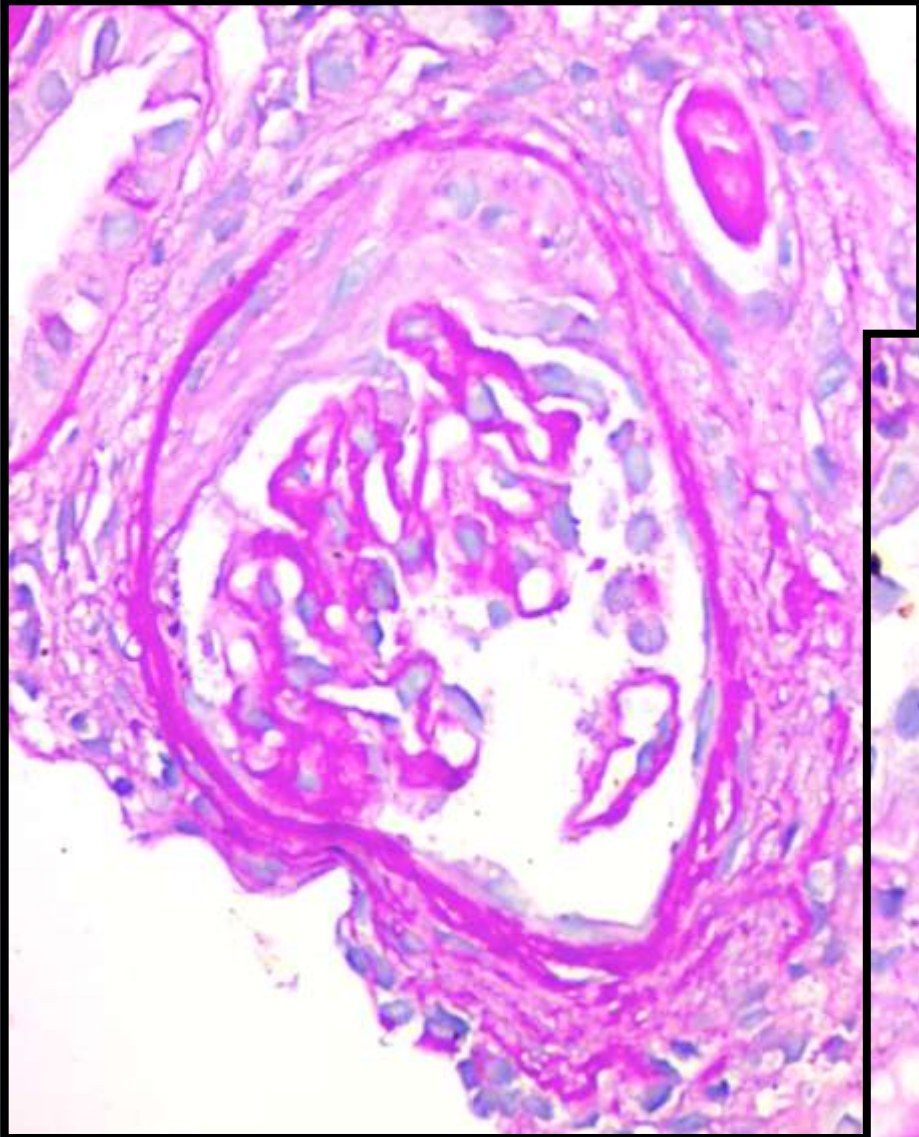
Hypertensive Nephropathy

“Benign Nephrosclerosis”

- *Two additional types of glomerular changes:*
 - Ischemic glomeruli
 - Solidified glomeruli



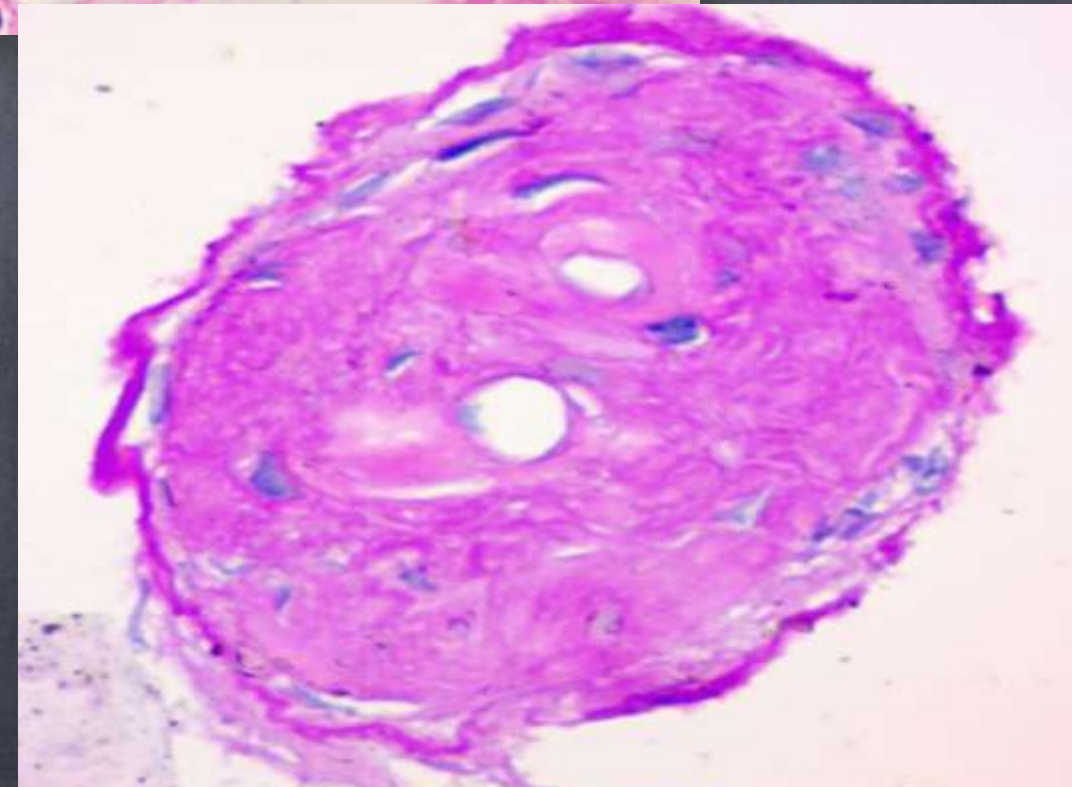
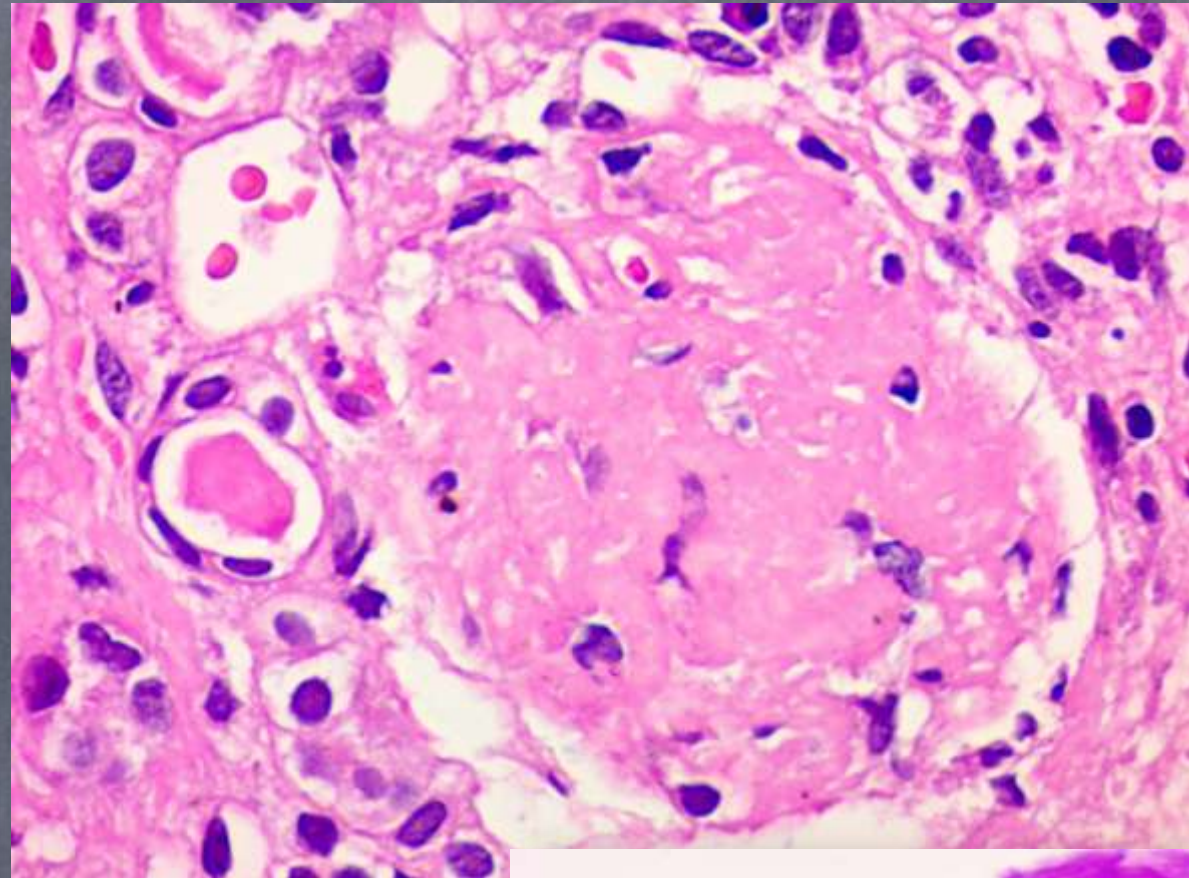




Hypertensive Nephropathy

“Benign Nephrosclerosis”

The solidified glomerulus is characterized by an increase in the mesangial matrix, which results in either segmental or global solidification (sclerosis) of the glomerular tuft extending to the Bowman capsule without collagenization of the Bowman space



Hypertensive Nephropathy

“Benign Nephrosclerosis”

□ Such glomeruli frequently contain hyalinosis lesions and represent “decompensated benign nephrosclerosis” (DBN)

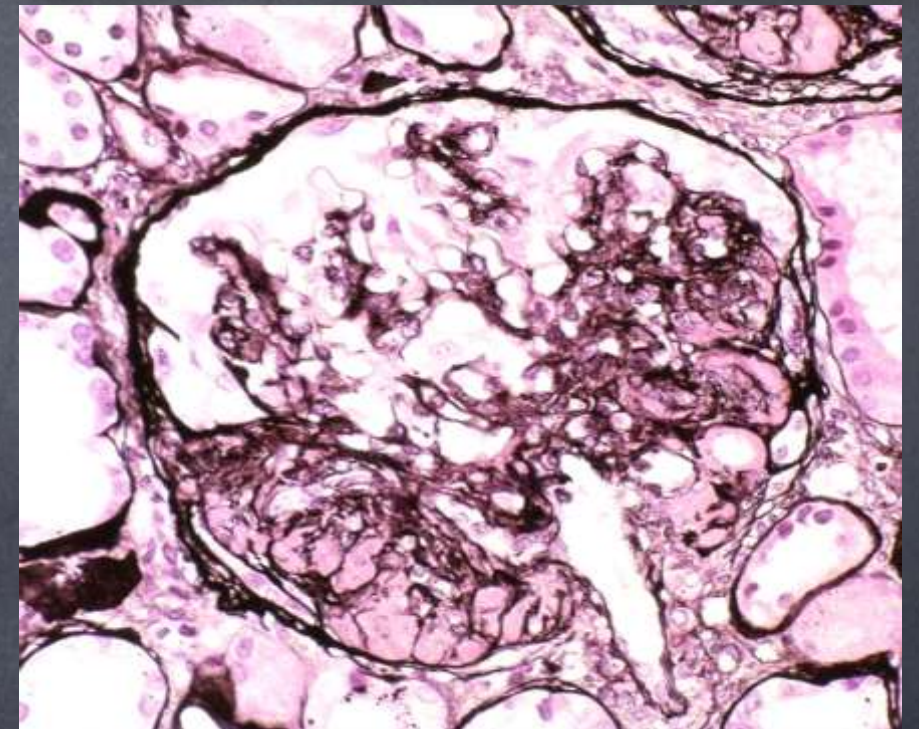
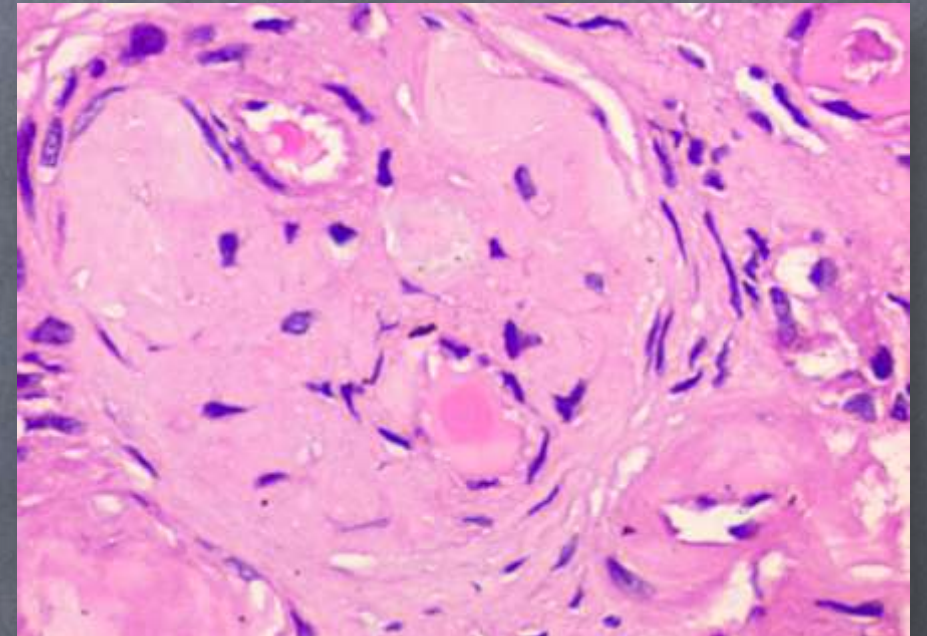
□ DBN was used by Bohle et al. in their study of 1177 patients and correlated it to

- chronic renal failure within a few years

- higher levels of blood pressure, proteinuria, and higher serum creatinine than patients with solely ischemic lesions

□ Others found them more in African americans and were associated by DBN

□ May represent a Secondary form of FSGS



Bohle A, et al. Kidney Int 1998;54(Suppl 67):S205
Marcantoni C et al. Kidney Int 2002;62:172

Hypertensive Nephropathy

“Benign Nephrosclerosis”

Other Changes

Hypertrophied glomeruli

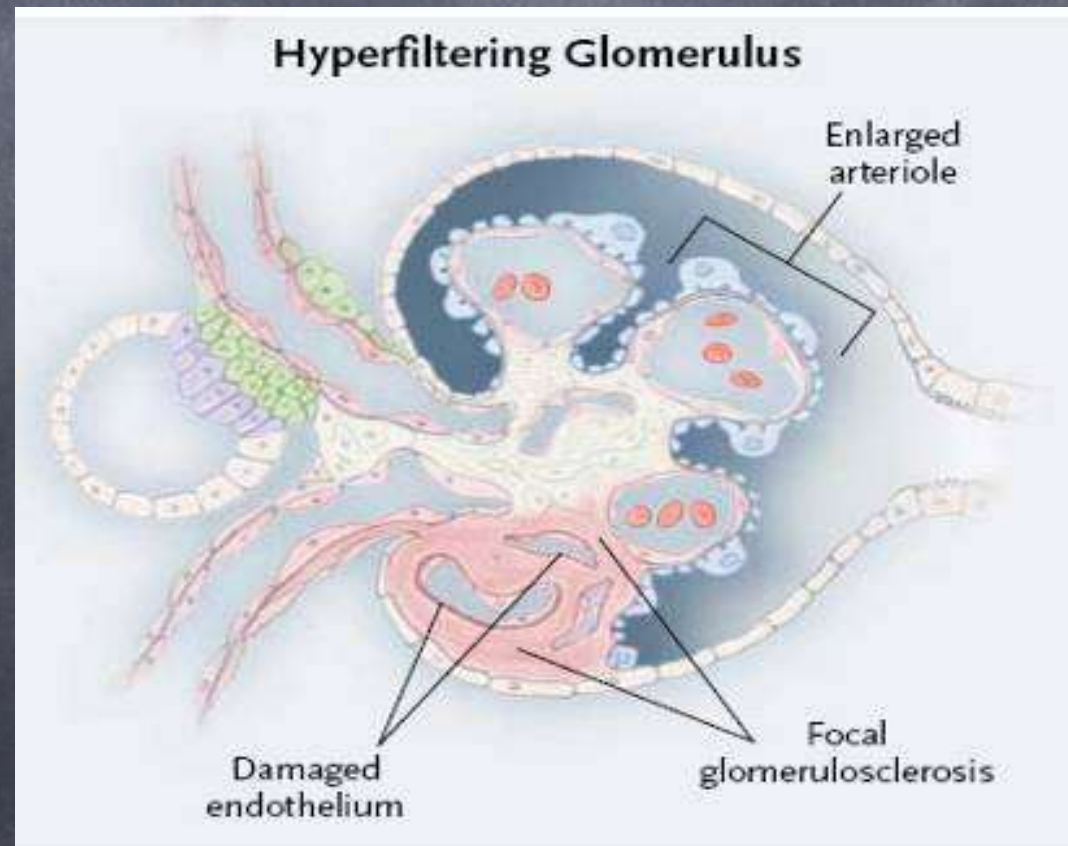
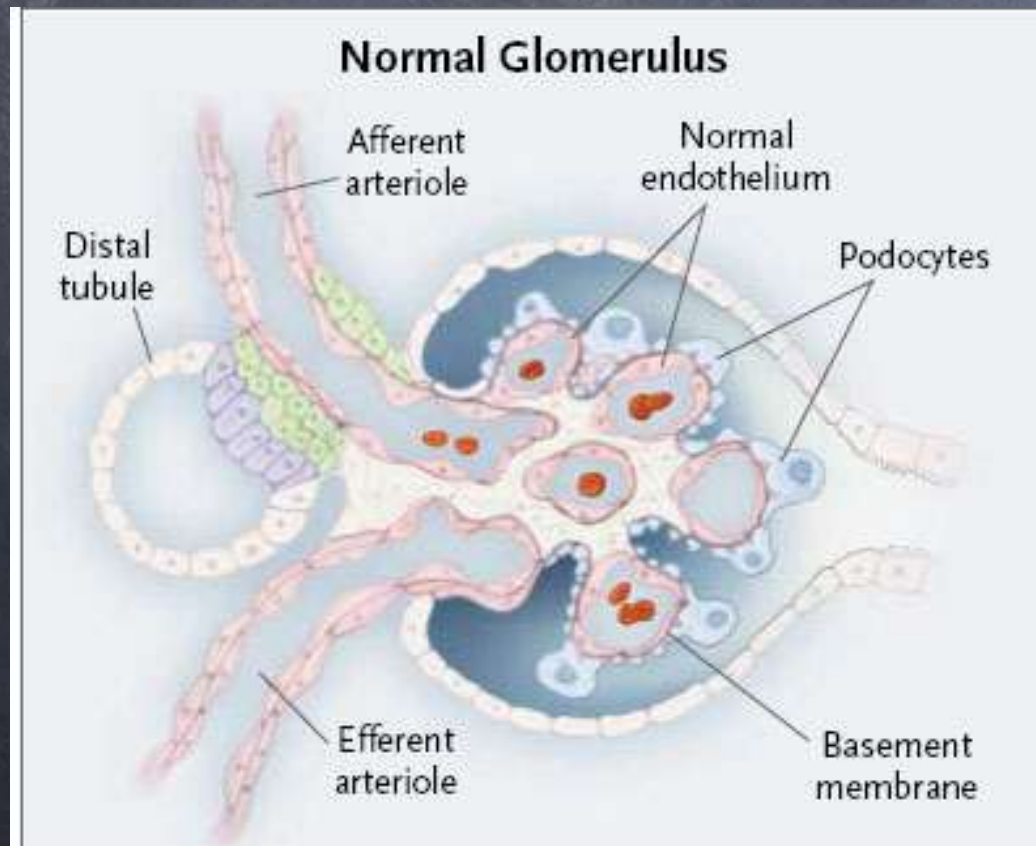
Decreased podocyte number

Lower glomerular number and mean glomerular volume

Hill GS et al. Kidney Int 2006;69:823

The presence of relatively few glomeruli leads to increased filtration by each glomerulus. Over time this hyperfiltration may cause injury

Ingelfinger J, NEJM 2003



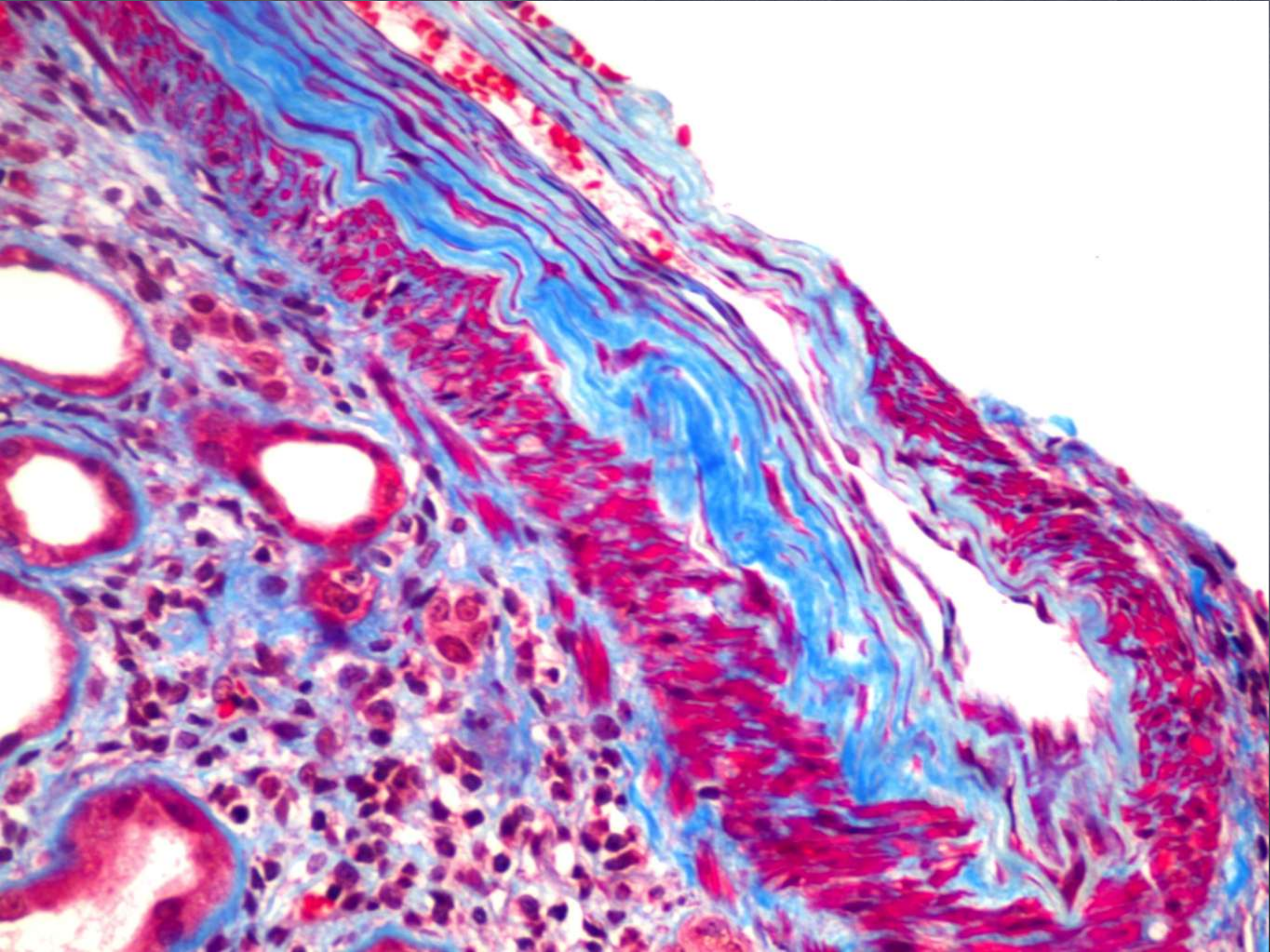
Hypertensive Nephropathy

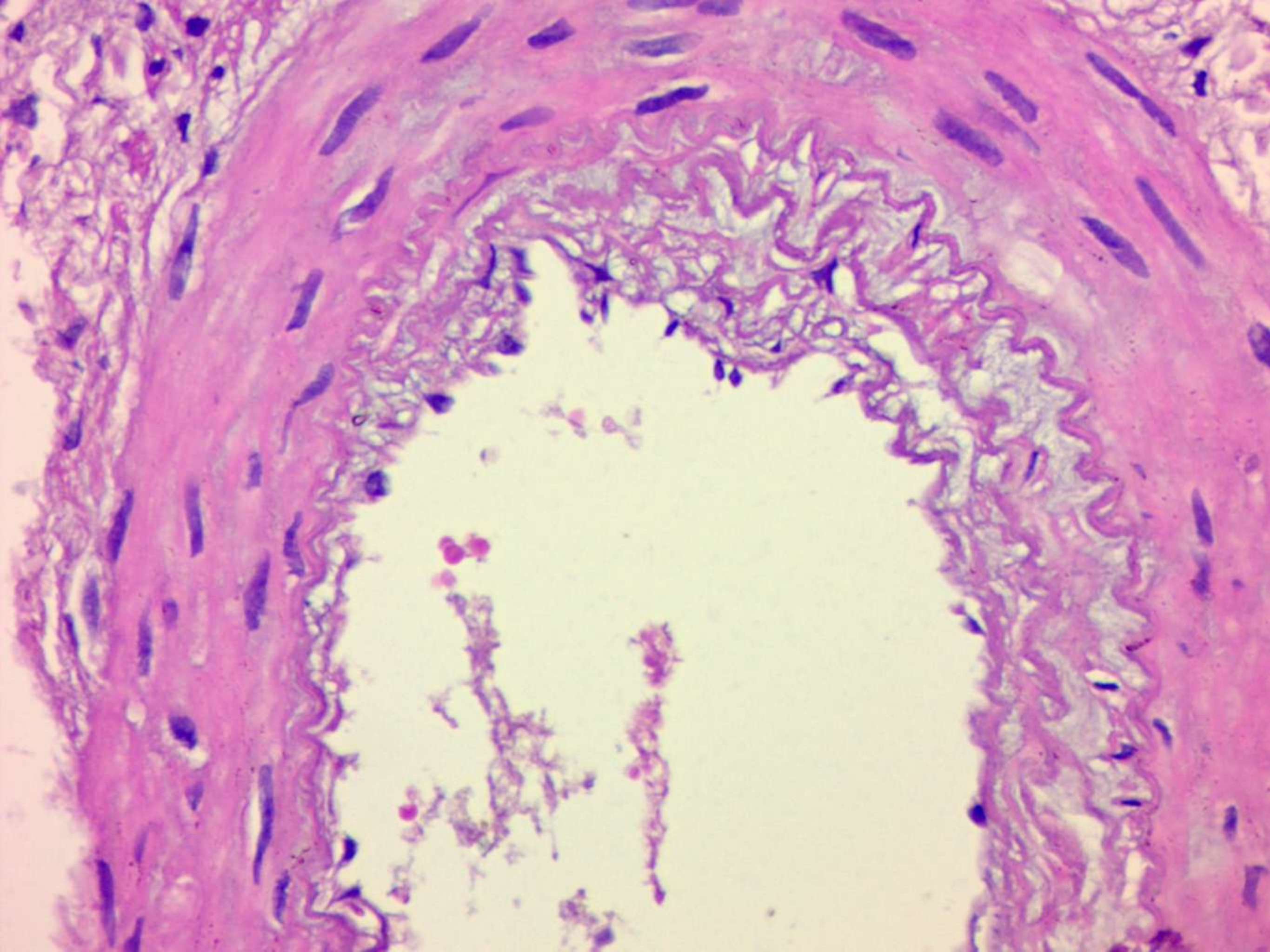
“Benign Nephrosclerosis”

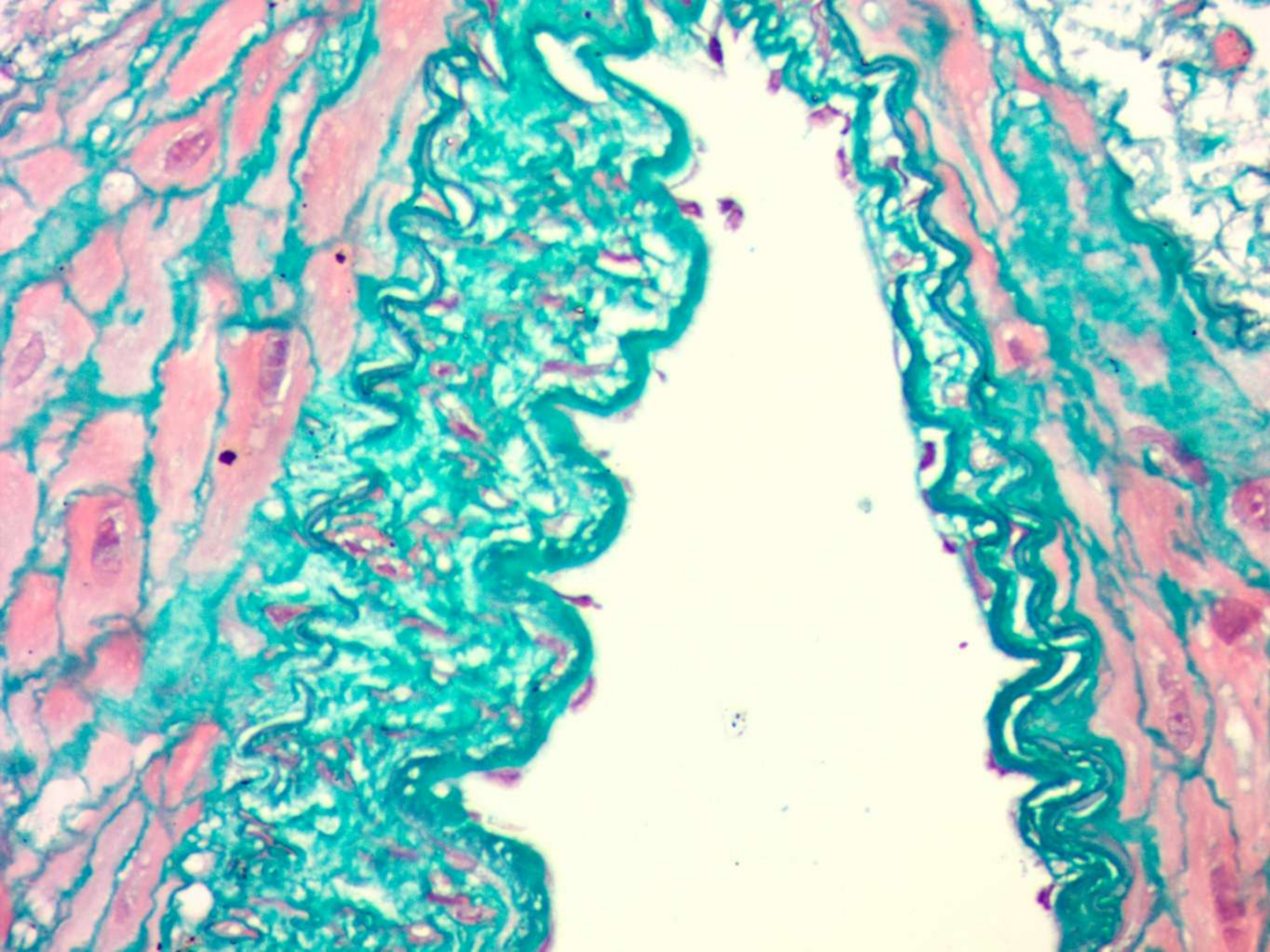
BLOOD VESSELS

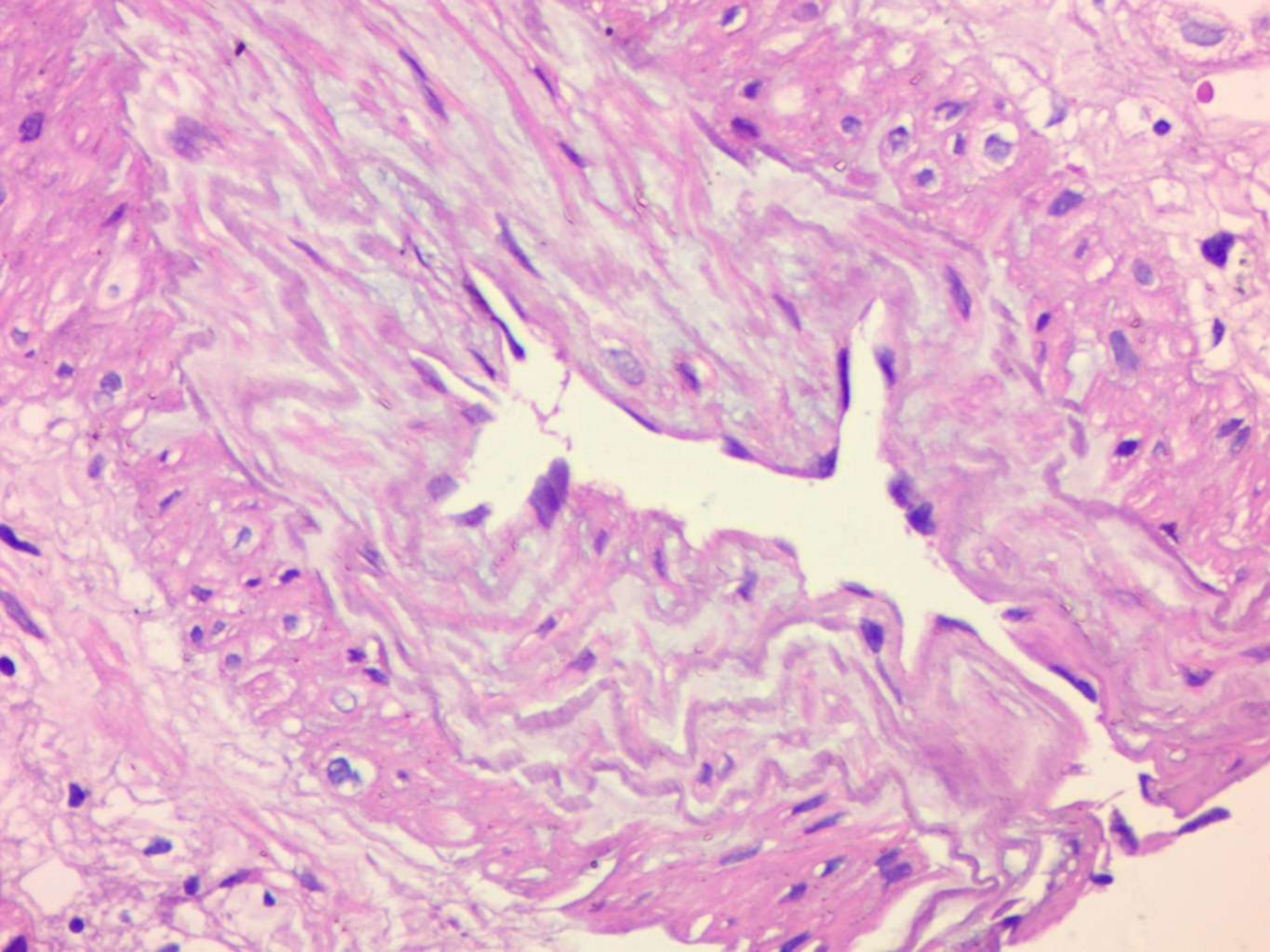
The changes vary with the size of the vessel involved and also differ among individual patients

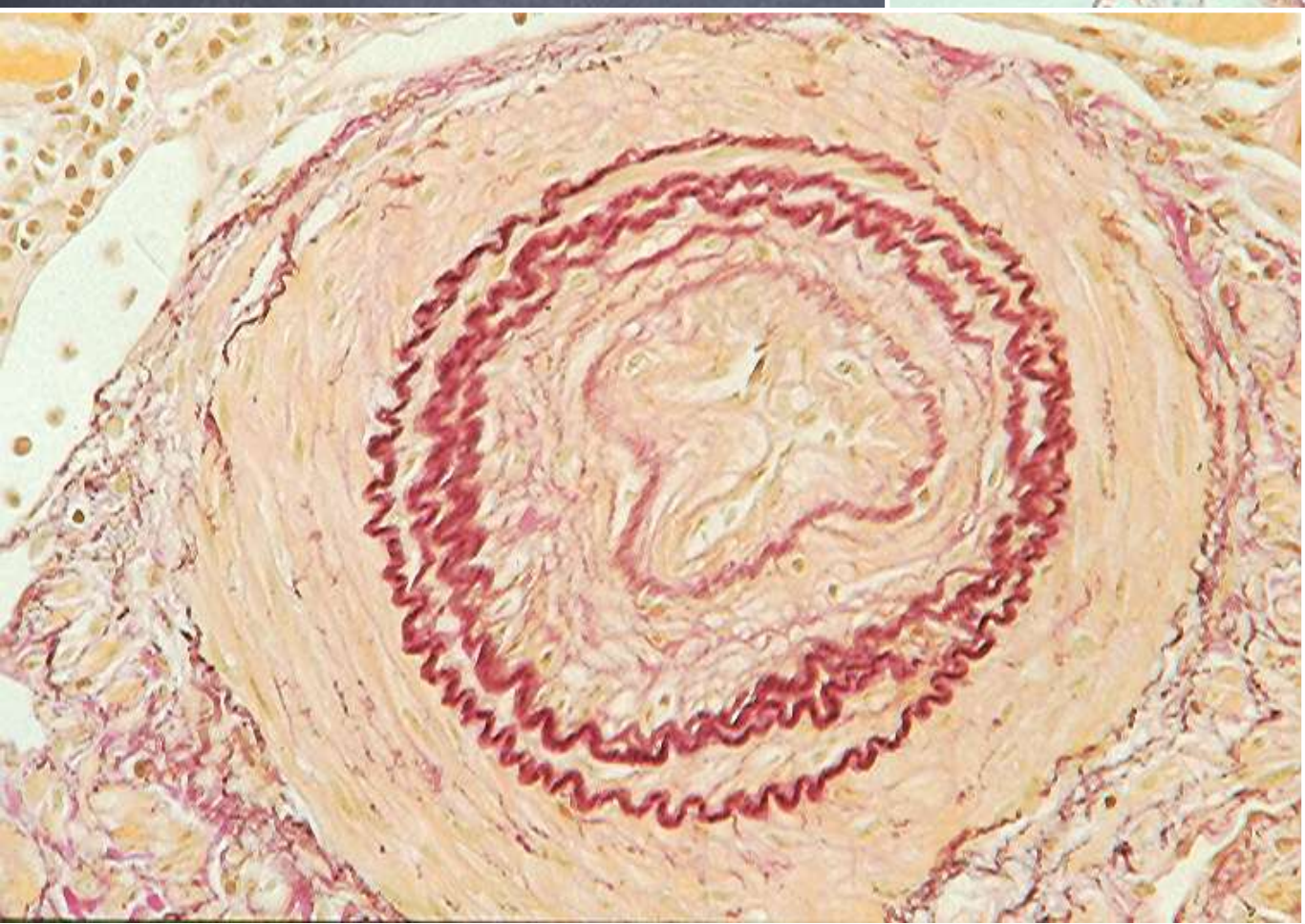
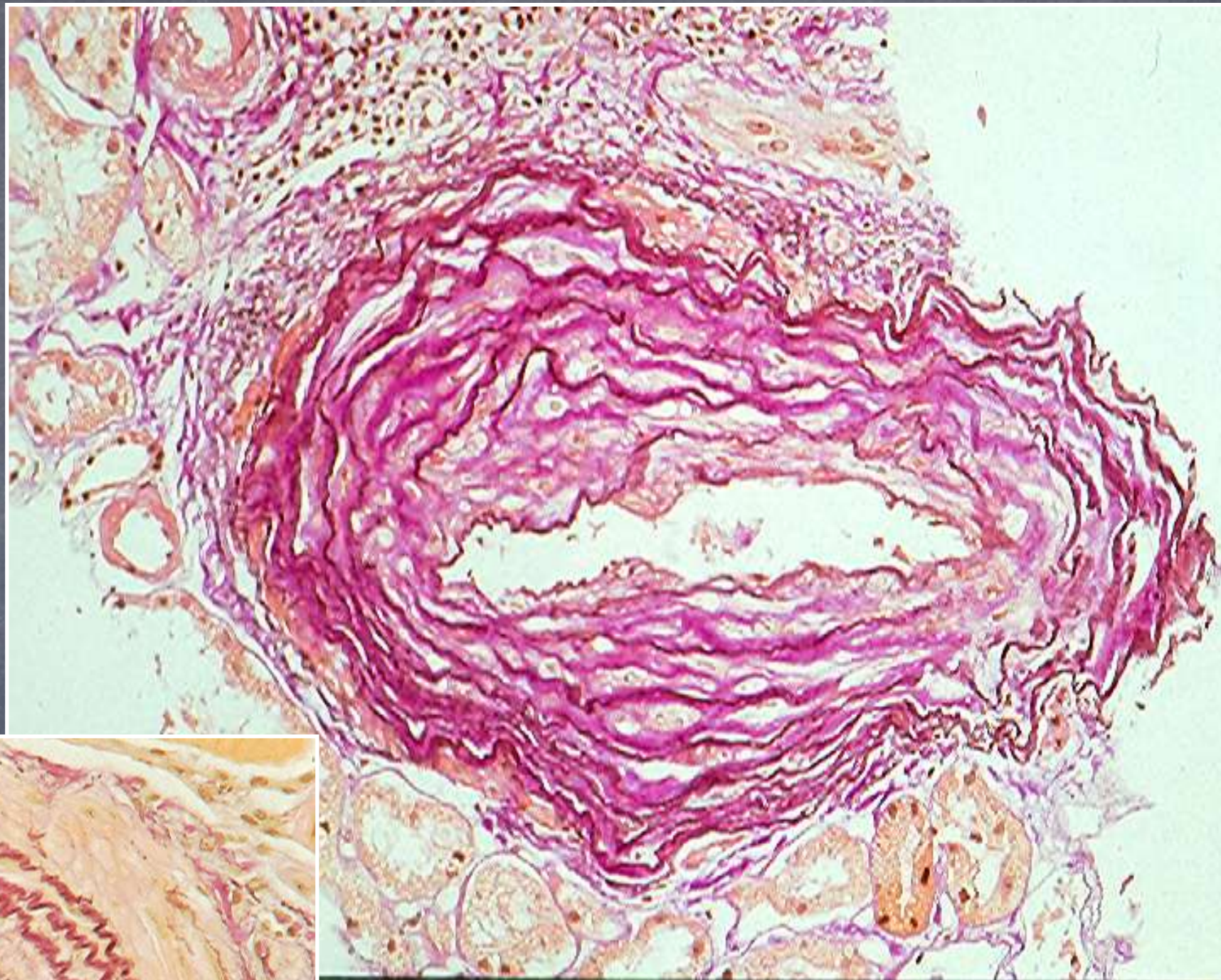
- ❑ Arcuate and larger arteries “atherosclerosis”
 - Fibrous intimal thickening with reduction of the vessel lumen
 - Maybe splitting of internal elastic lamina
- ❑ Interlobular arteries show fibroelastic intimal thickening with reduplication of the internal elastic lamina
- ❑ Arterioles show hyalinosis “*hyaline arteriolosclerosis*”
 - Risk increased with increases in diastolic blood pressure
 - The severity correlates with the degree of larger artery intimal thickening

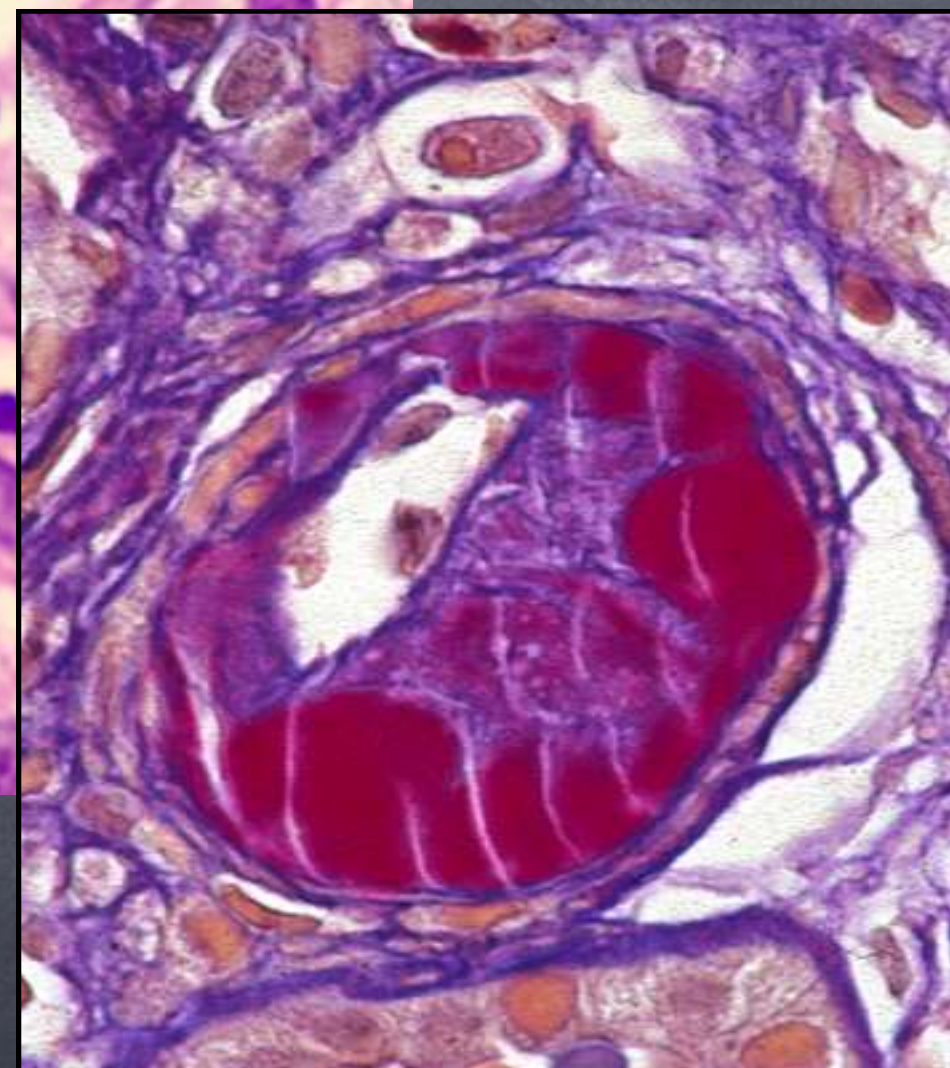
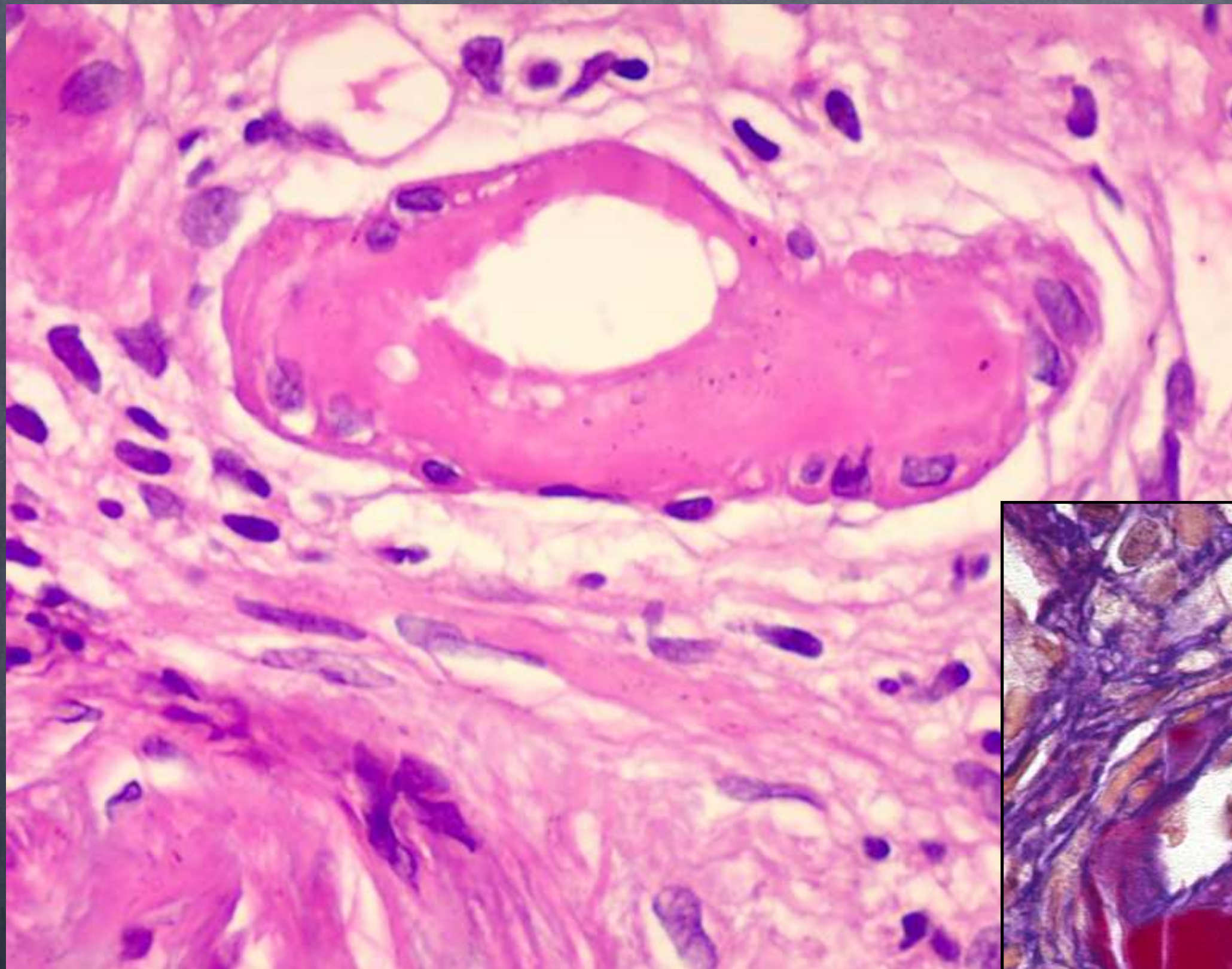






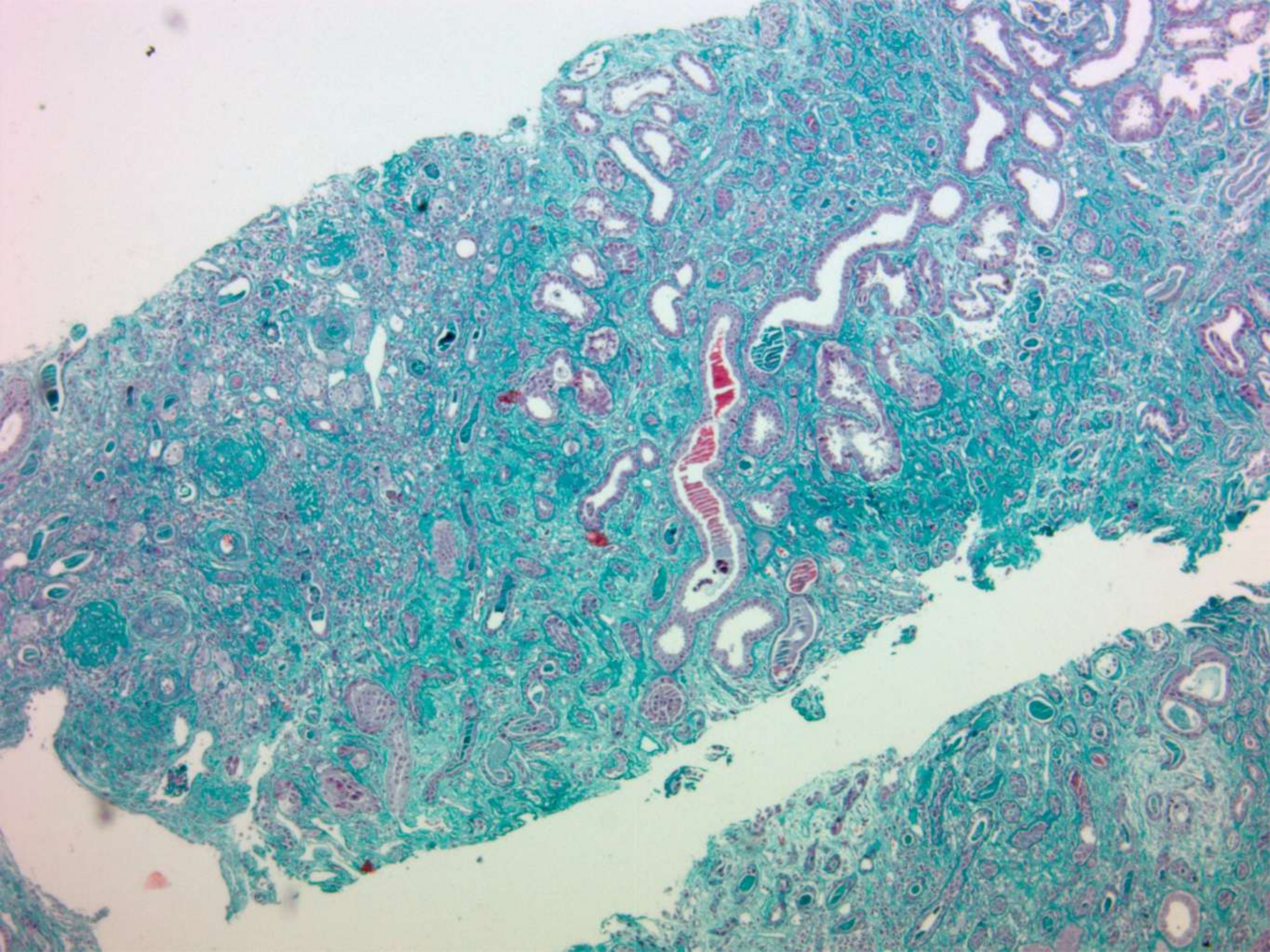


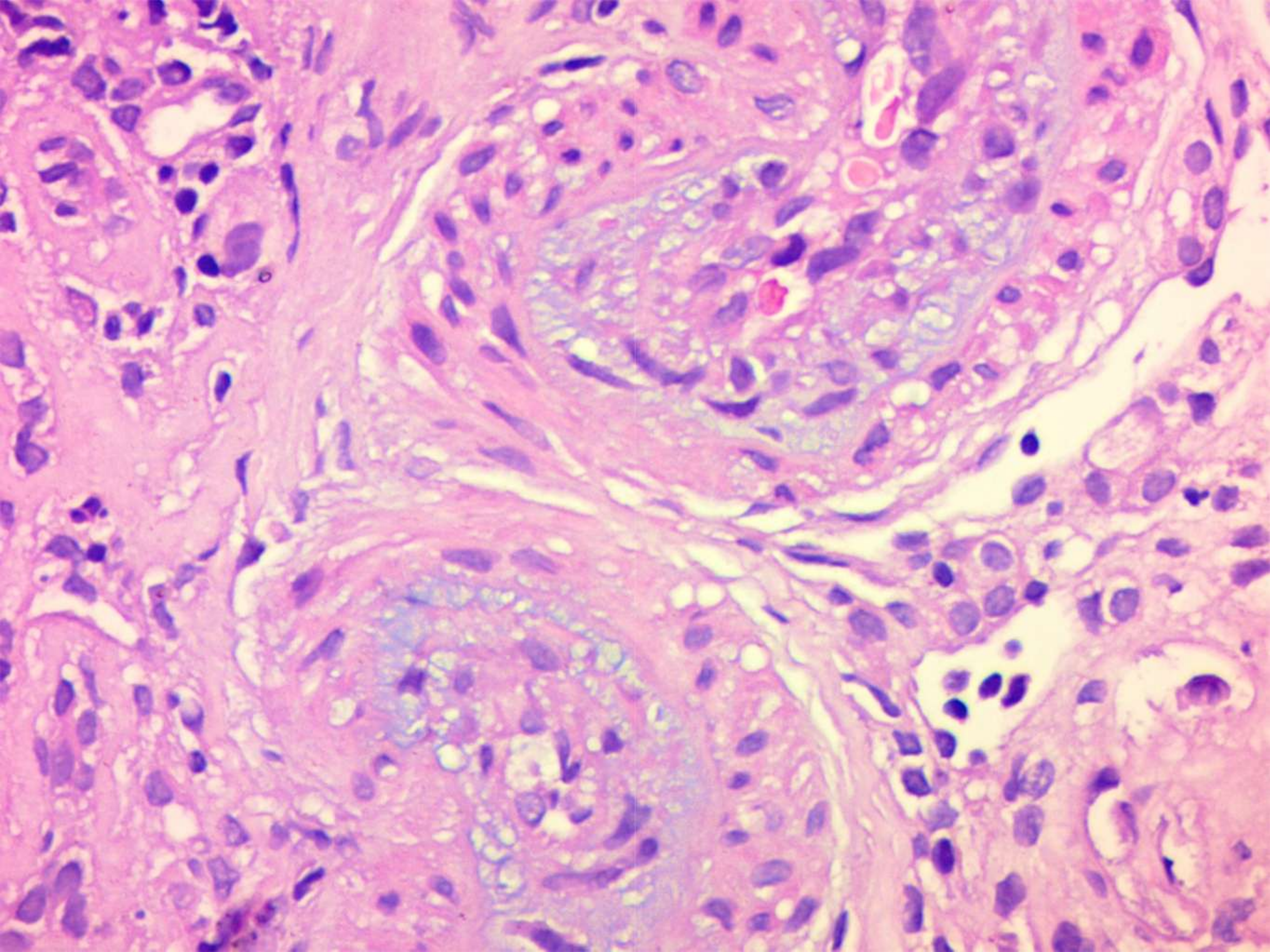


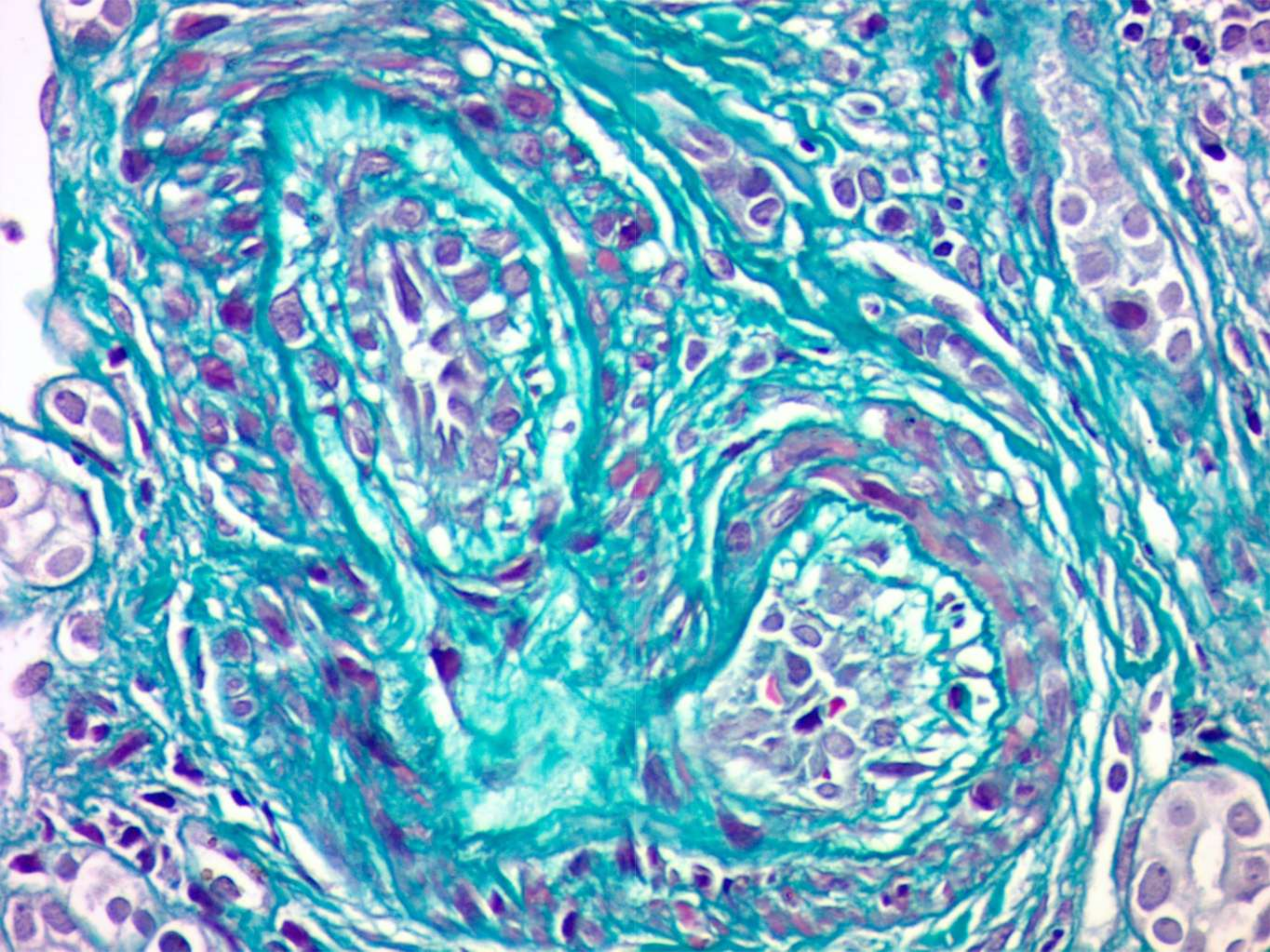


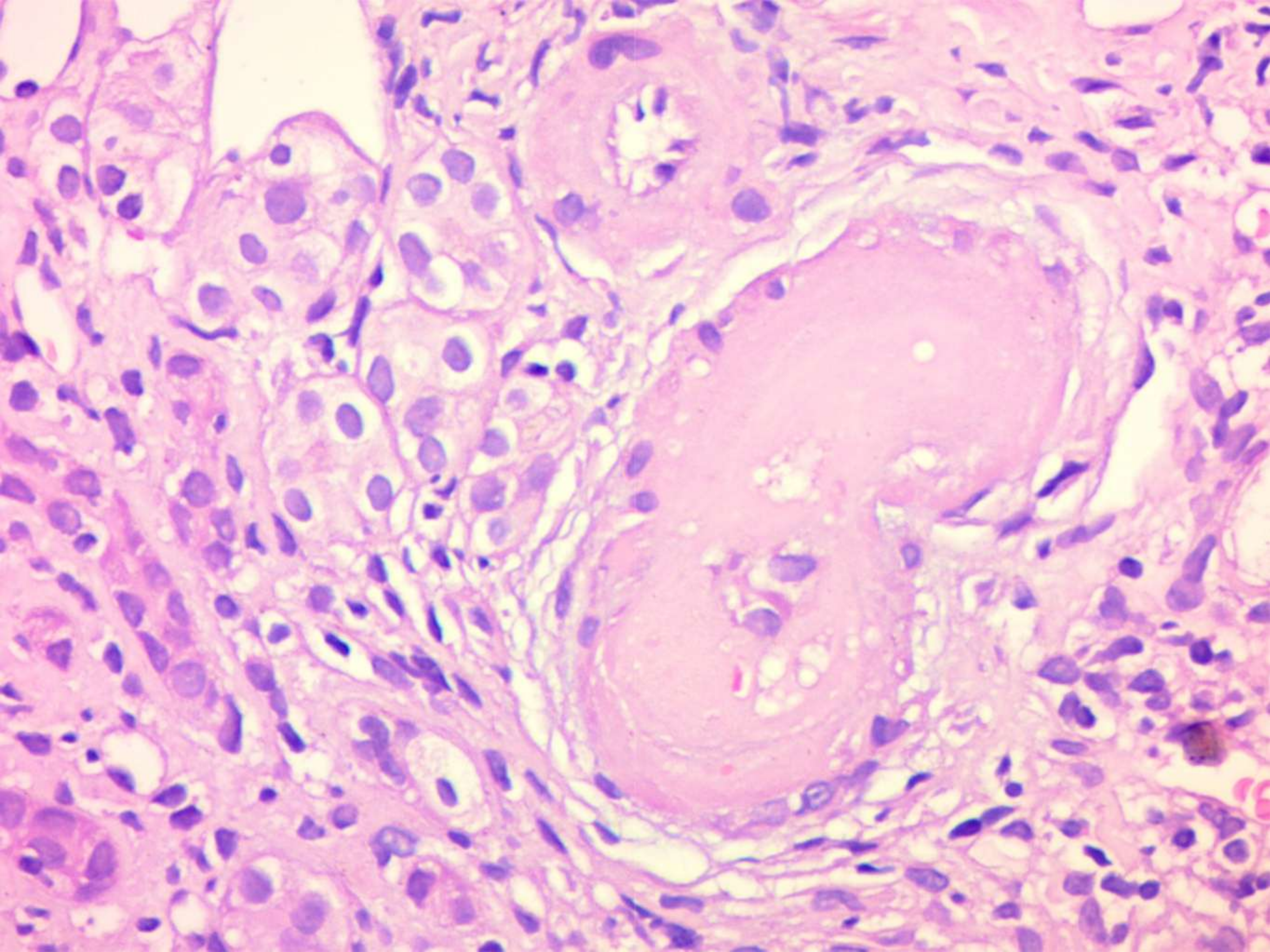
Case 1

- ❑ Male patient presented with epistaxis and serum creatinine 14 mg%
- ❑ On examination Bp was 230/140
- ❑ Metabolic acidosis, sAlb was 3.5, LDH 427, Na 135, K 3.6, Ca 7.8
- ❑ Urine analysis: RBCs 10-12, Pus 20-25, Alb ++, PCR: 3.92, Granular casts
- ❑ US: 11.2x4,8 and 9.8x4, duplex showed no renal artery stenosis
- ❑ Echo: EF 58%, Mild LVH
- ❑ Hemodialysis and renal biopsy

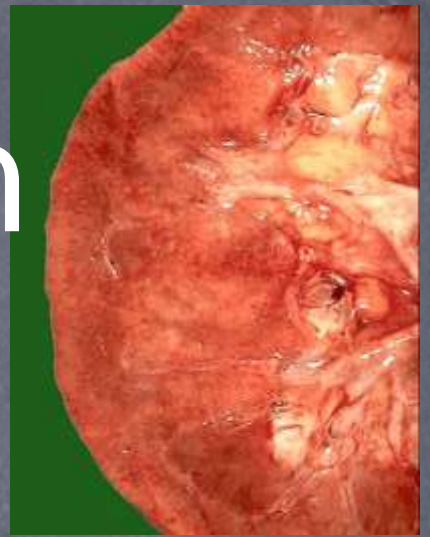




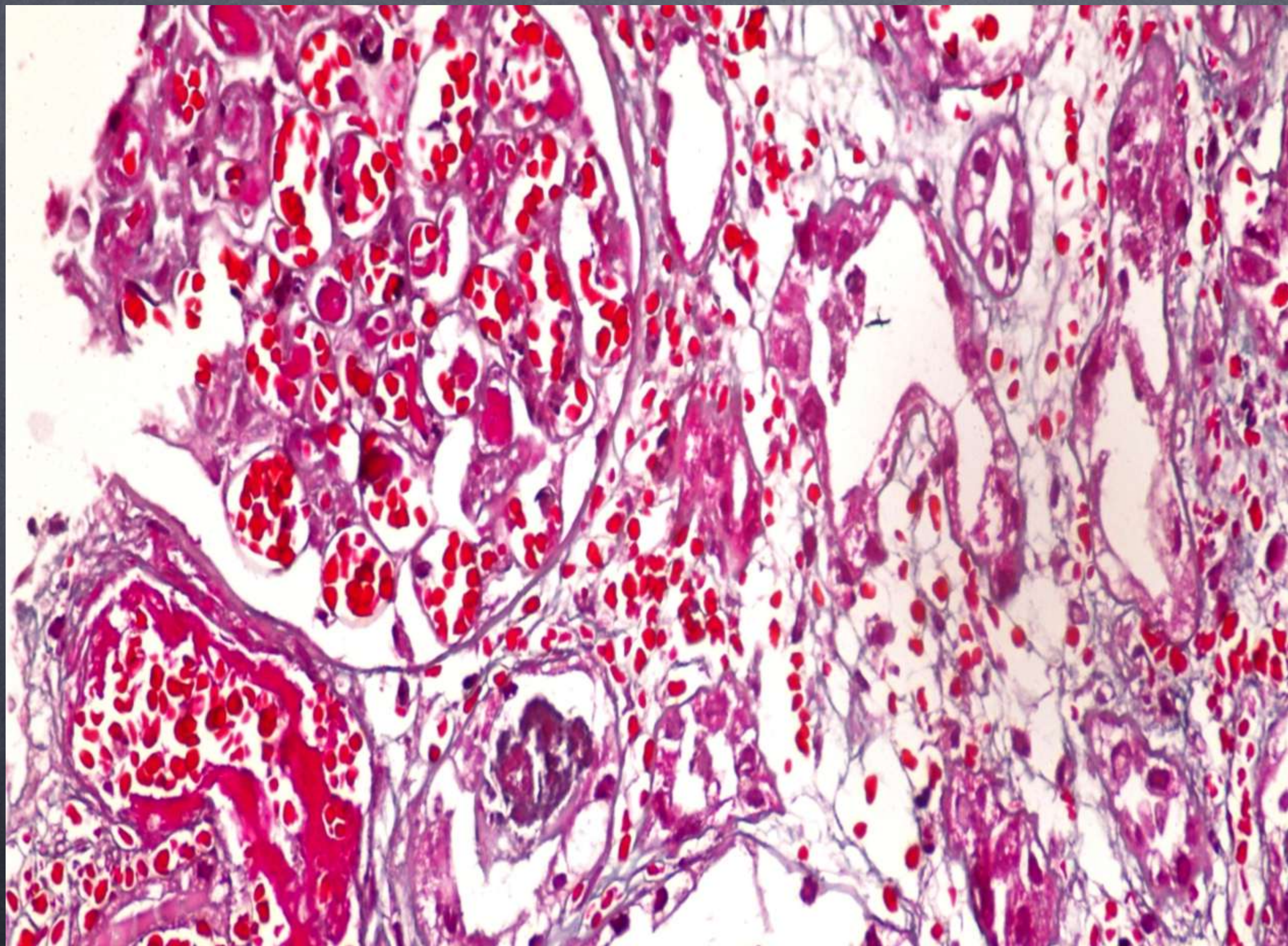


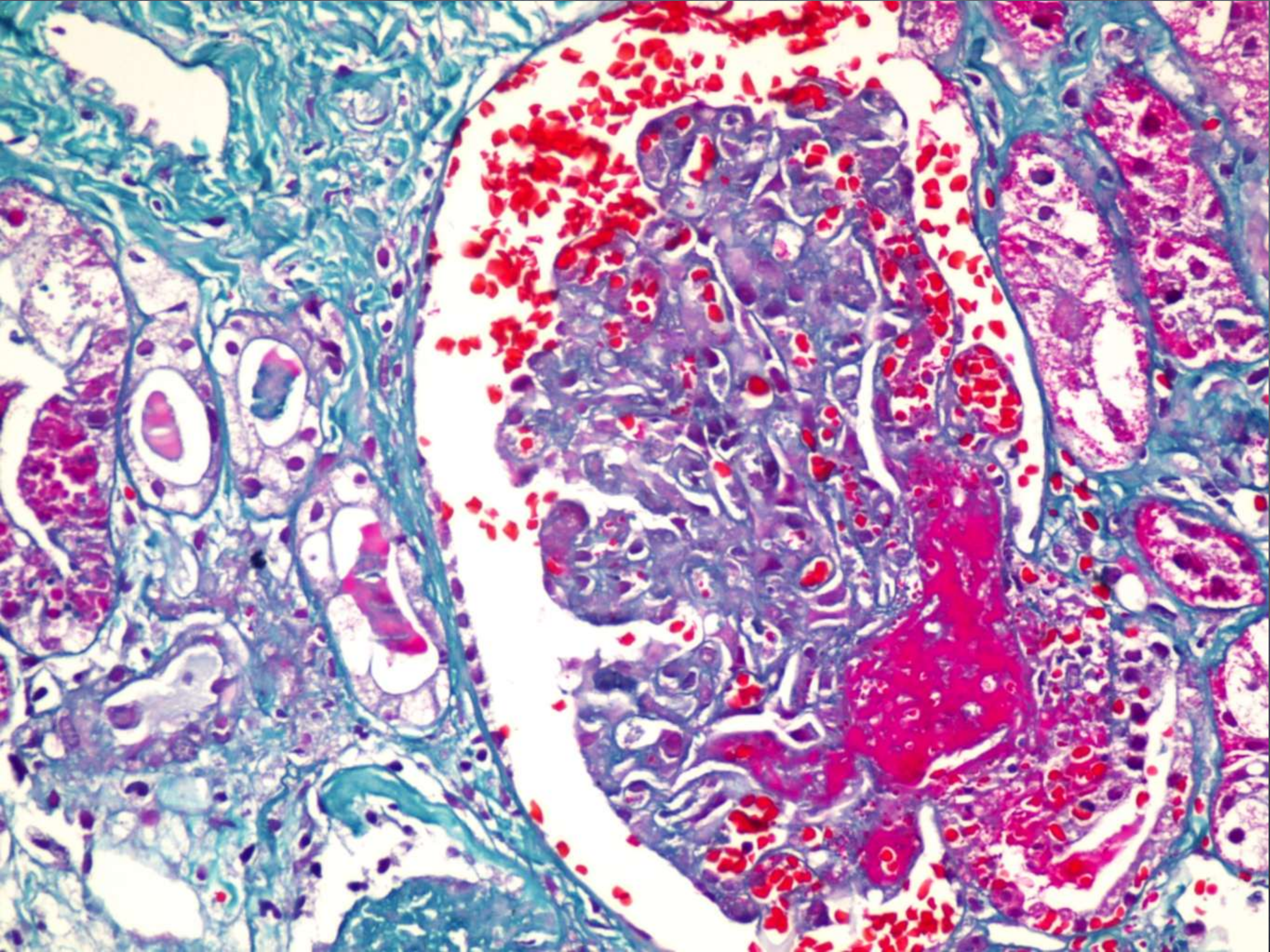


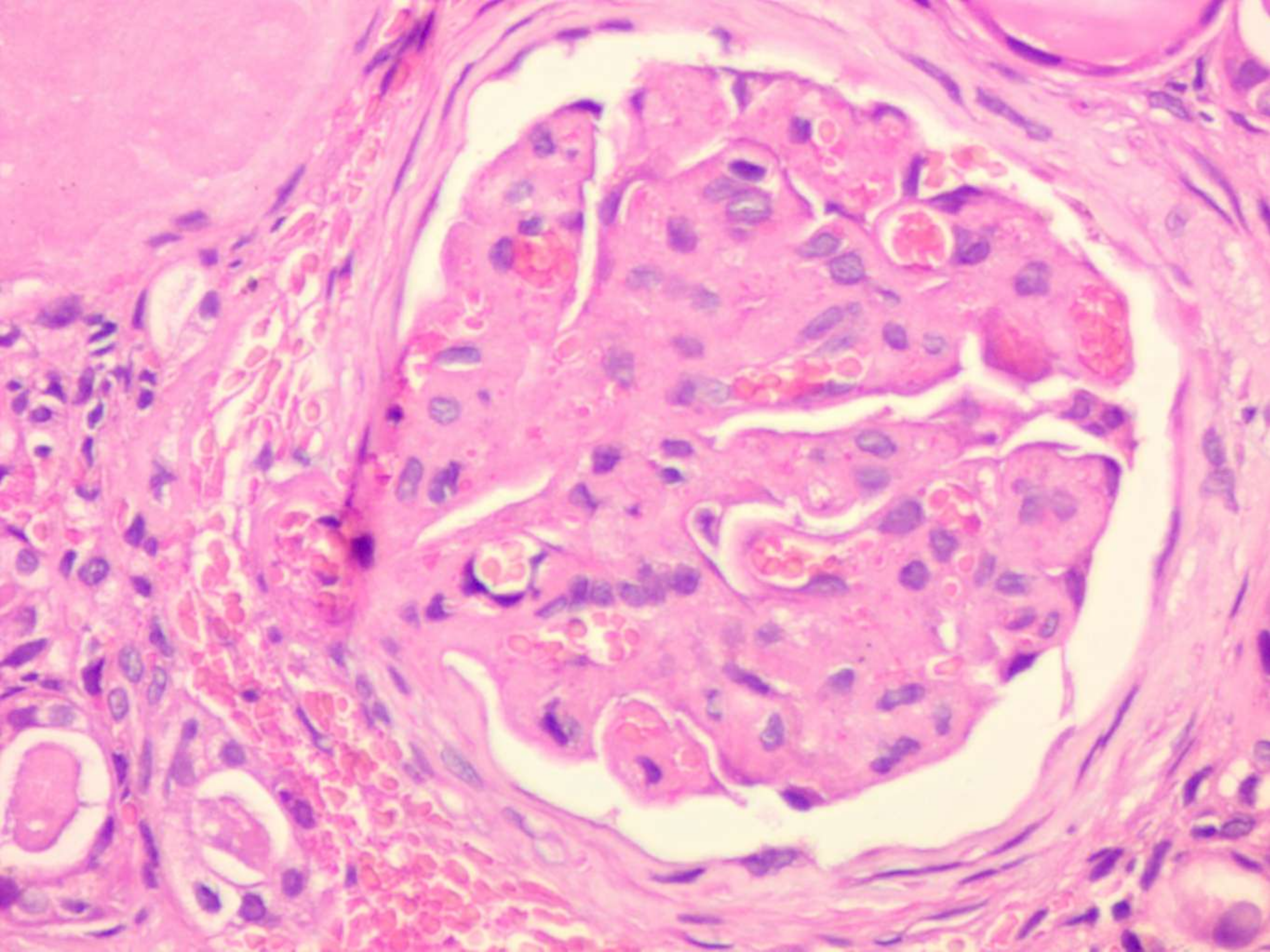
Accelerated Hypertension

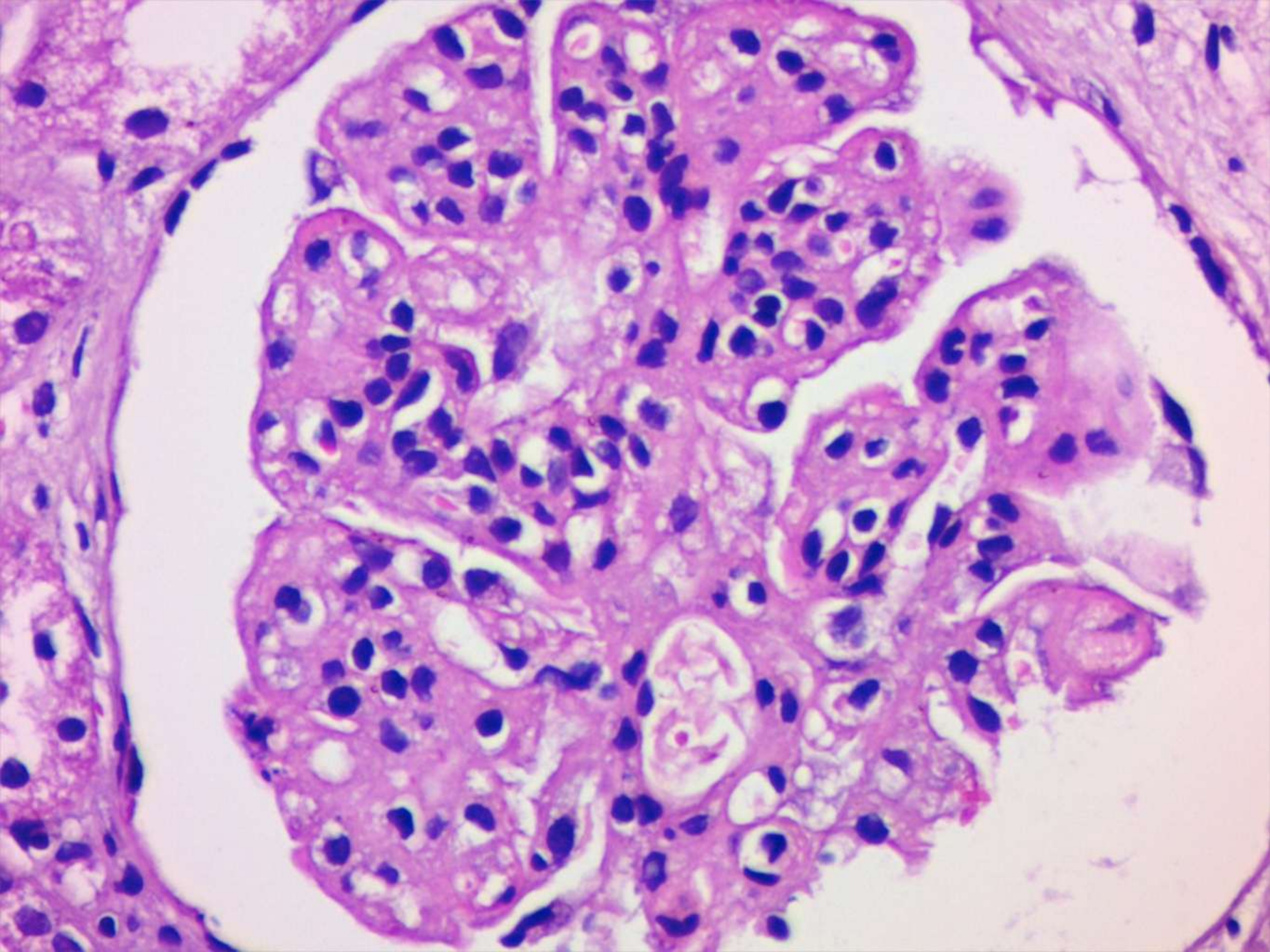


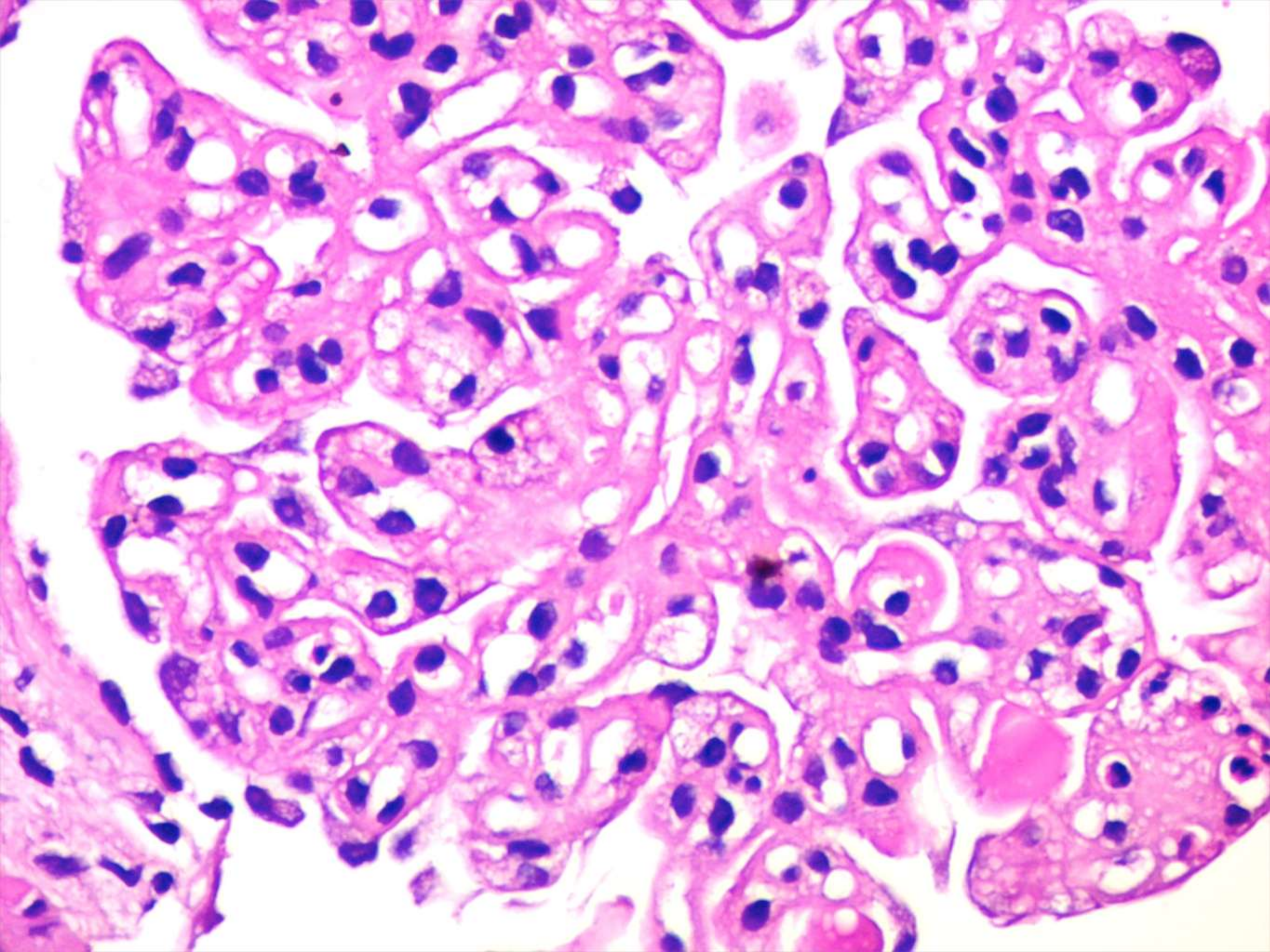
- ❑ “Malignant” hypertension is no longer used
- ❑ Can occur:
 - Without any history of hypertension
 - Maybe preceeded by essential hypertension
 - may complicate secondary forms or on a pre-existing renal disease
- ❑ The cause of the switch between benign to malignant is not fully understood (? renin-angiotensin)

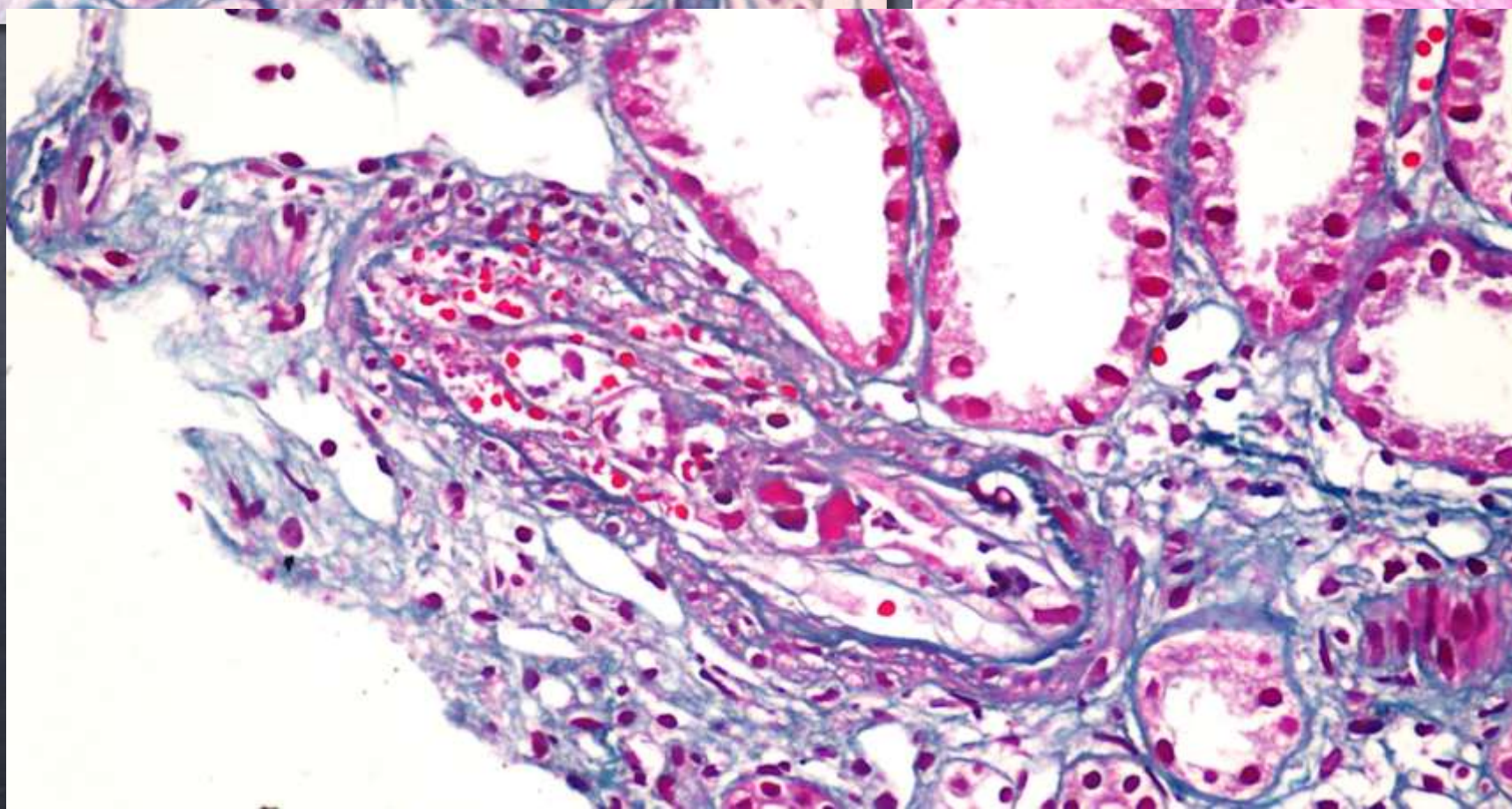
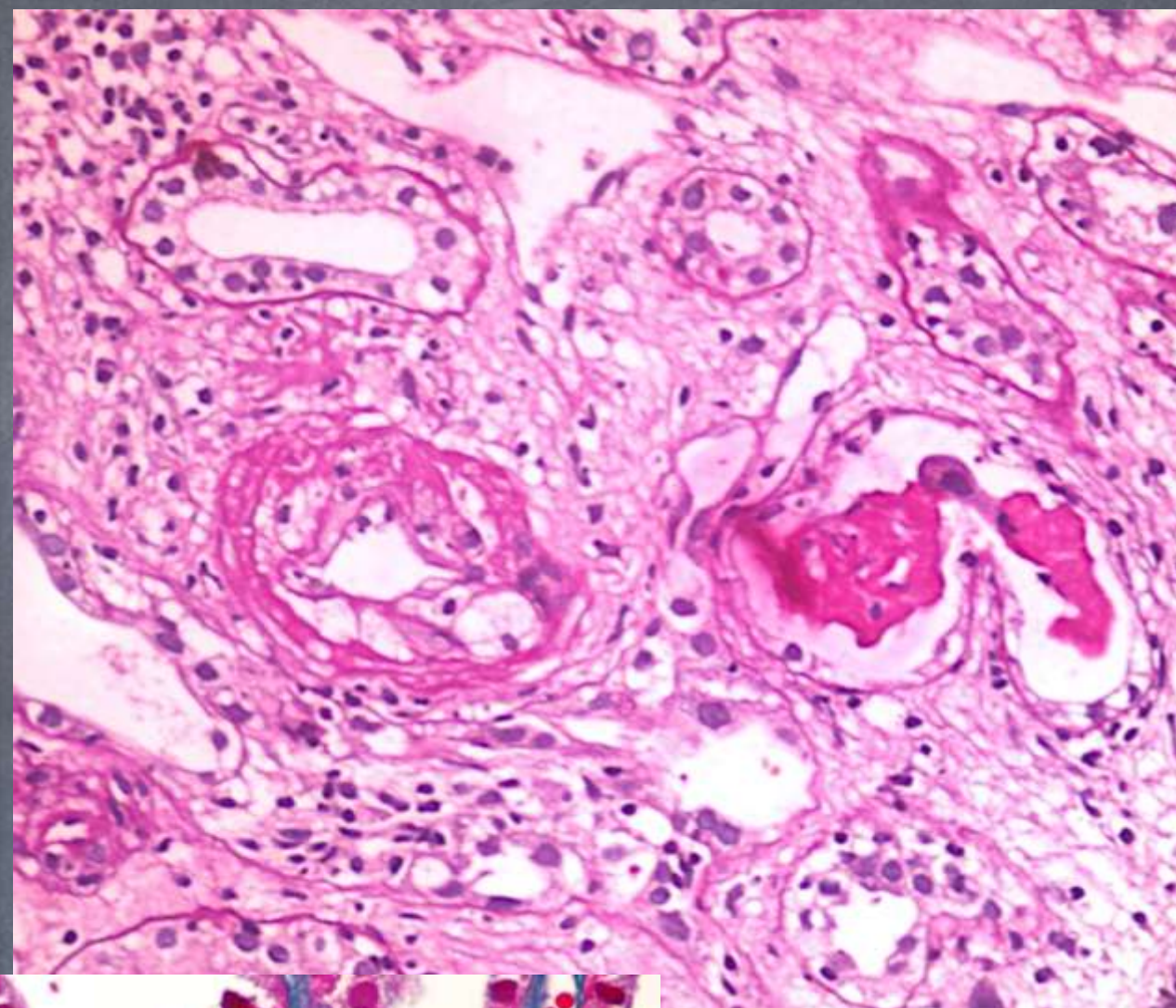
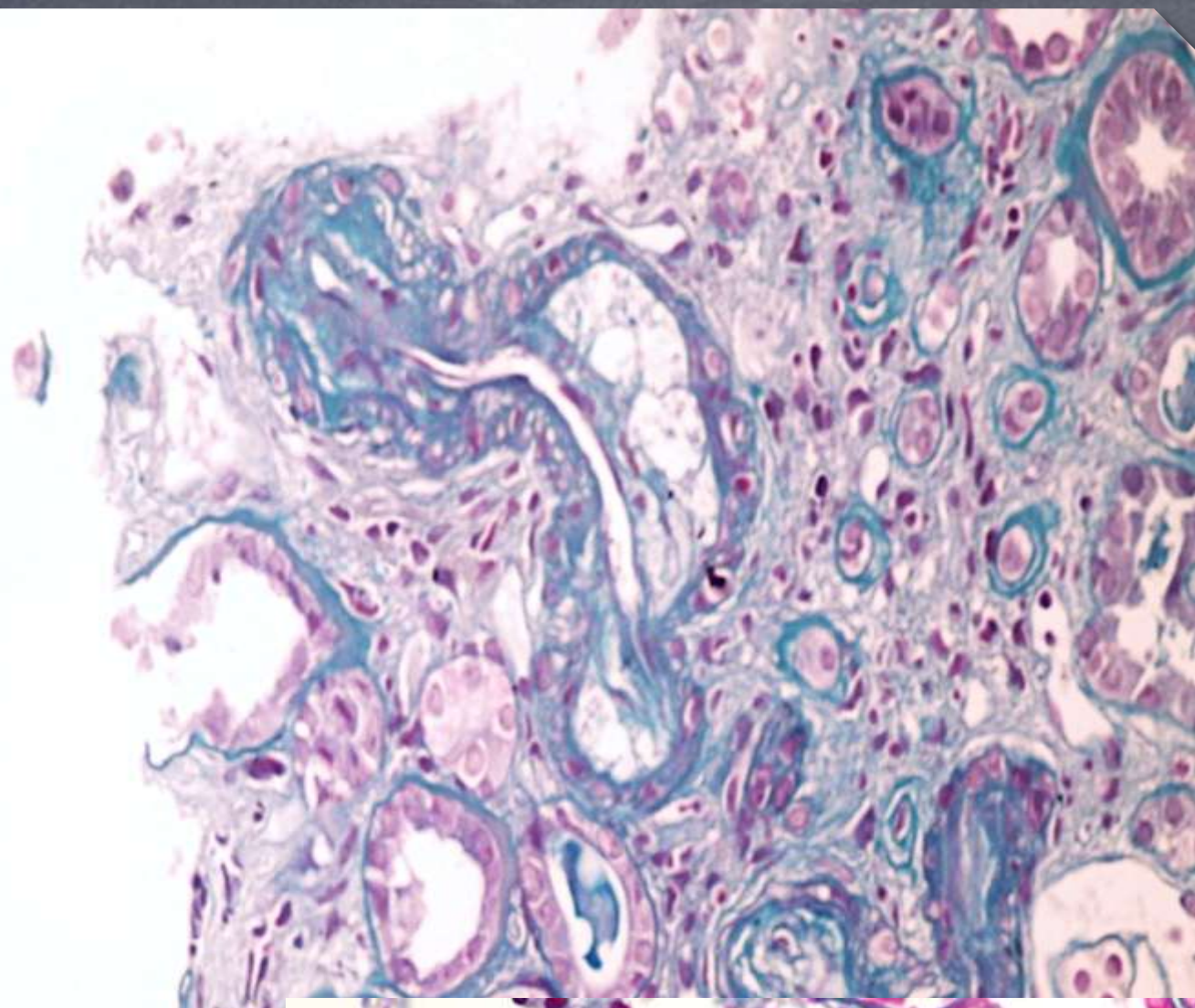


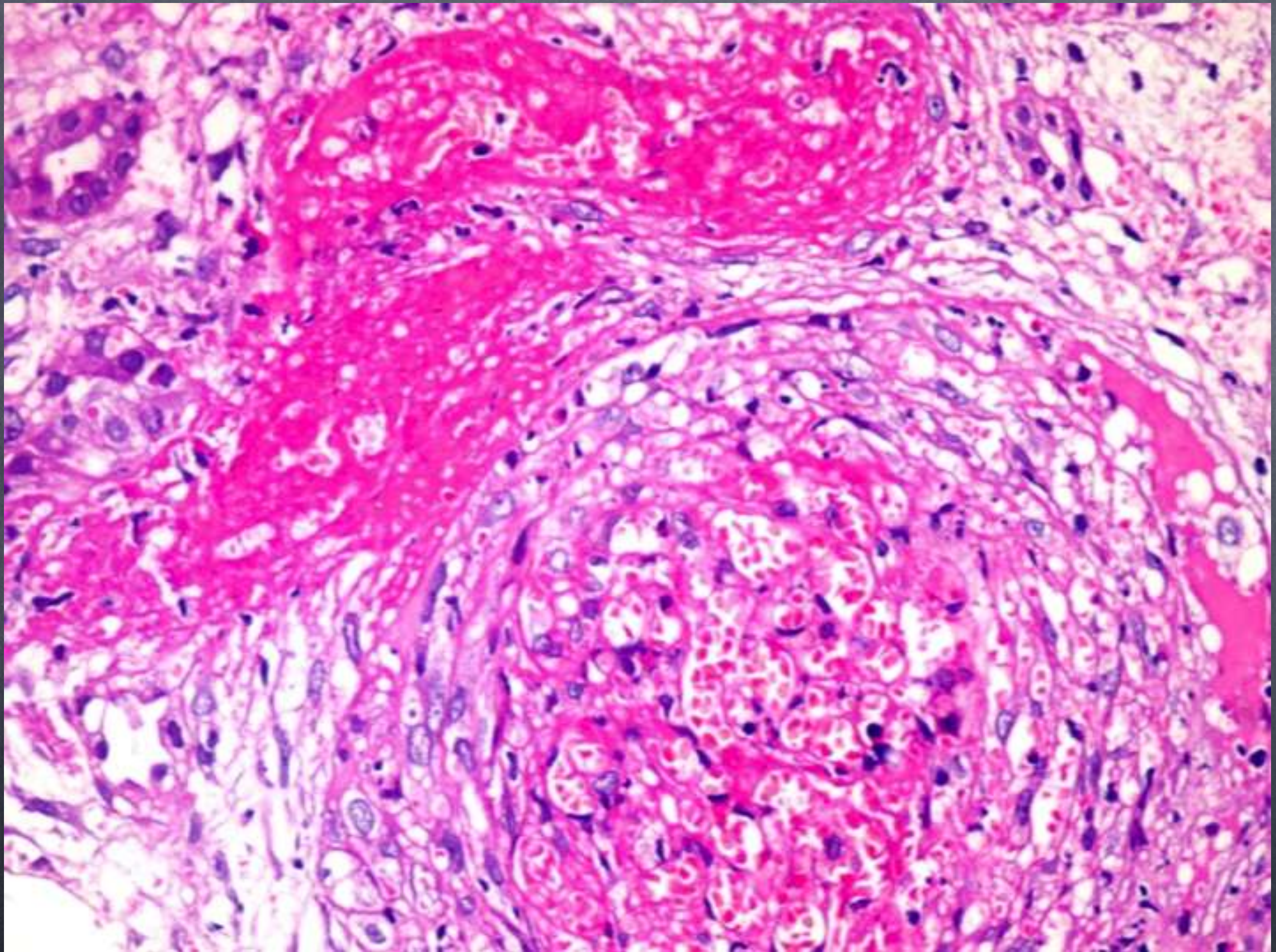


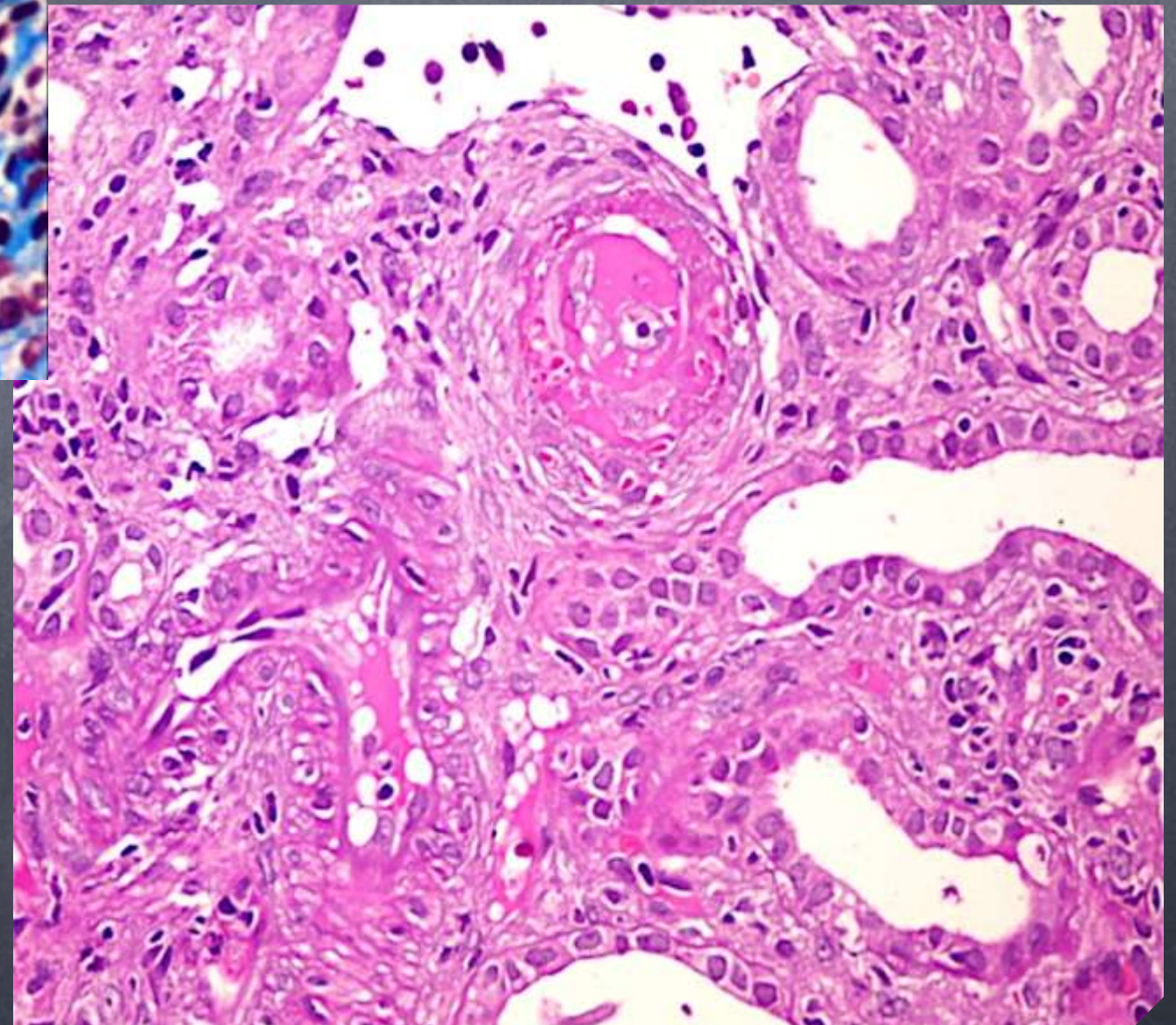
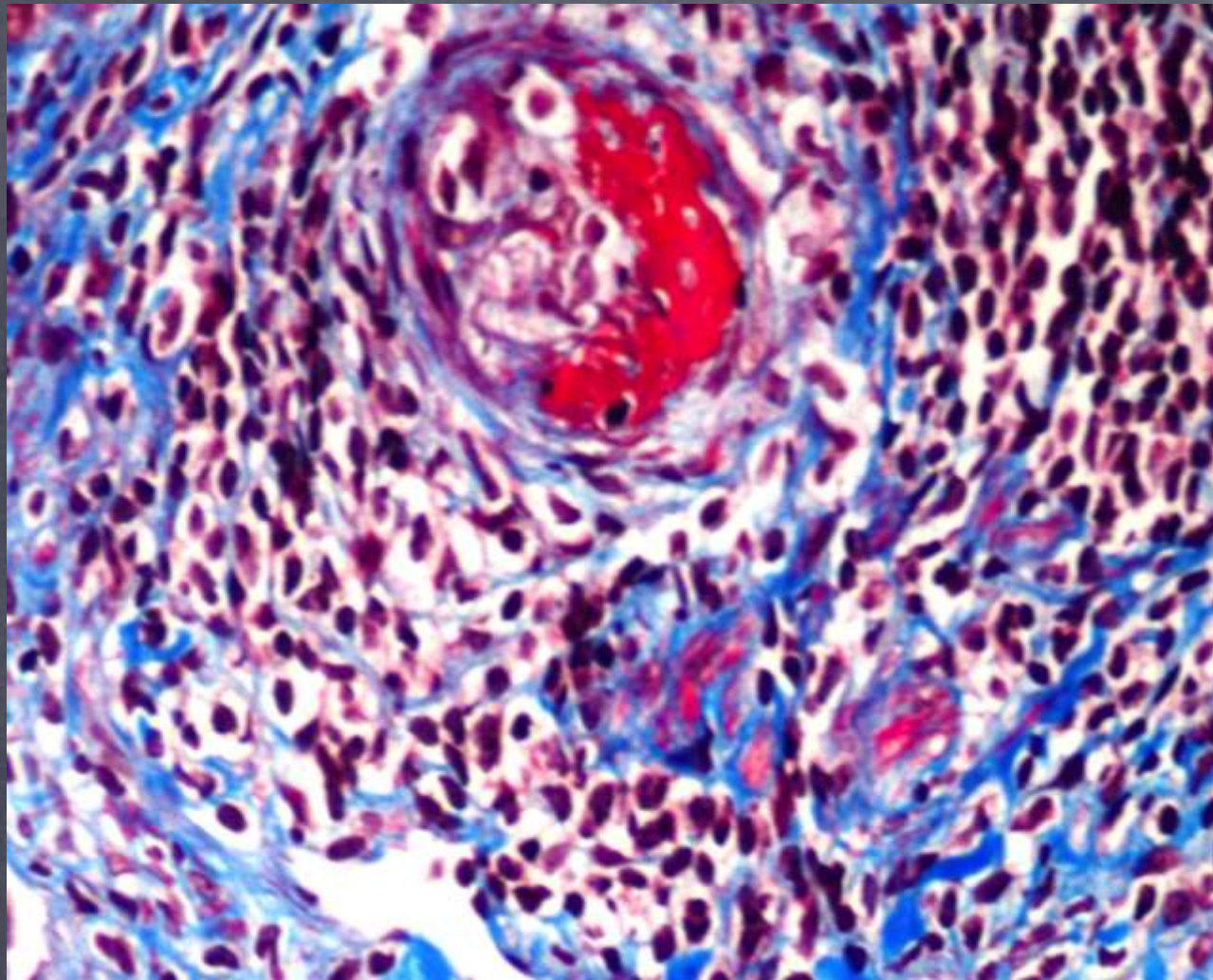


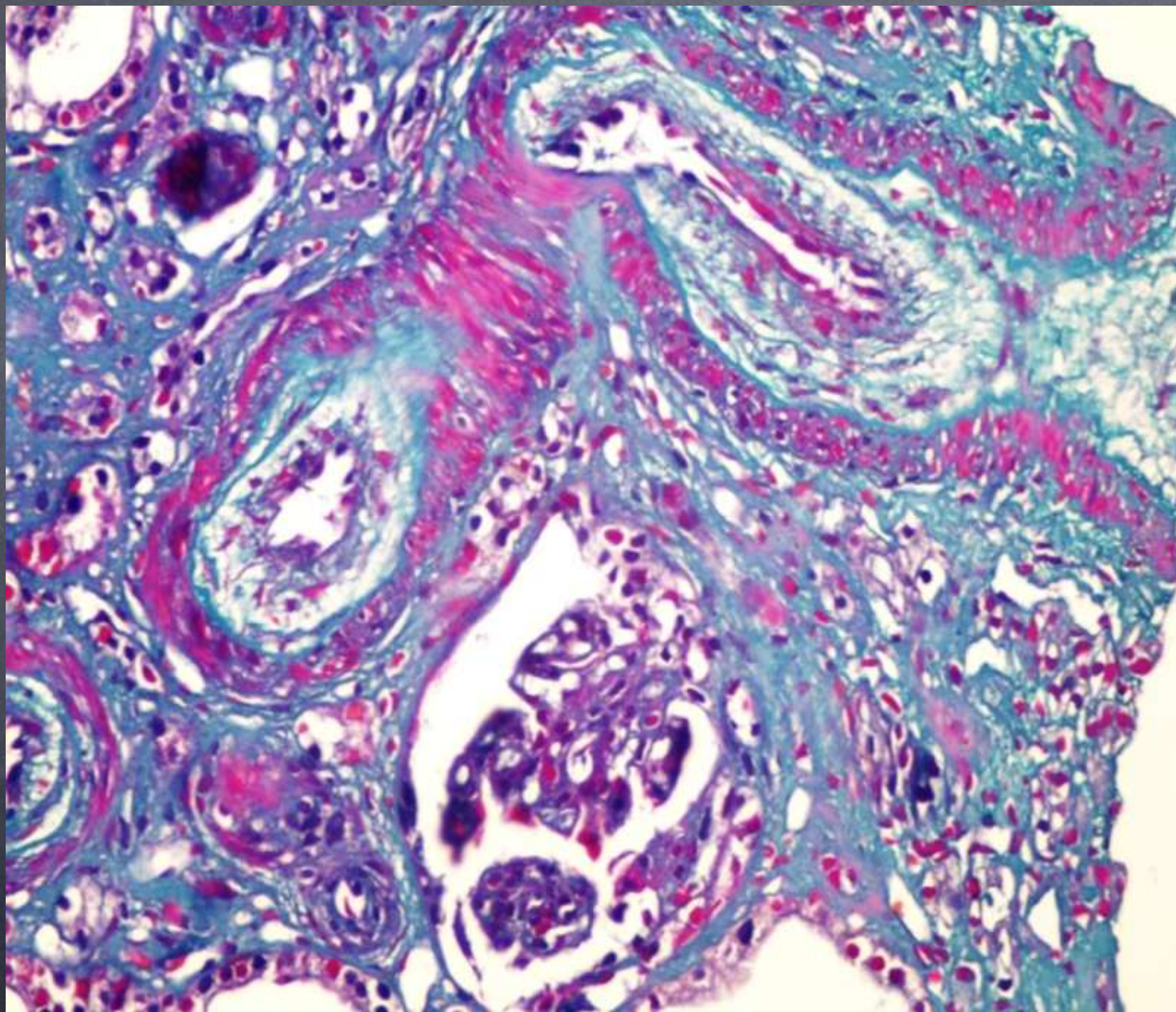


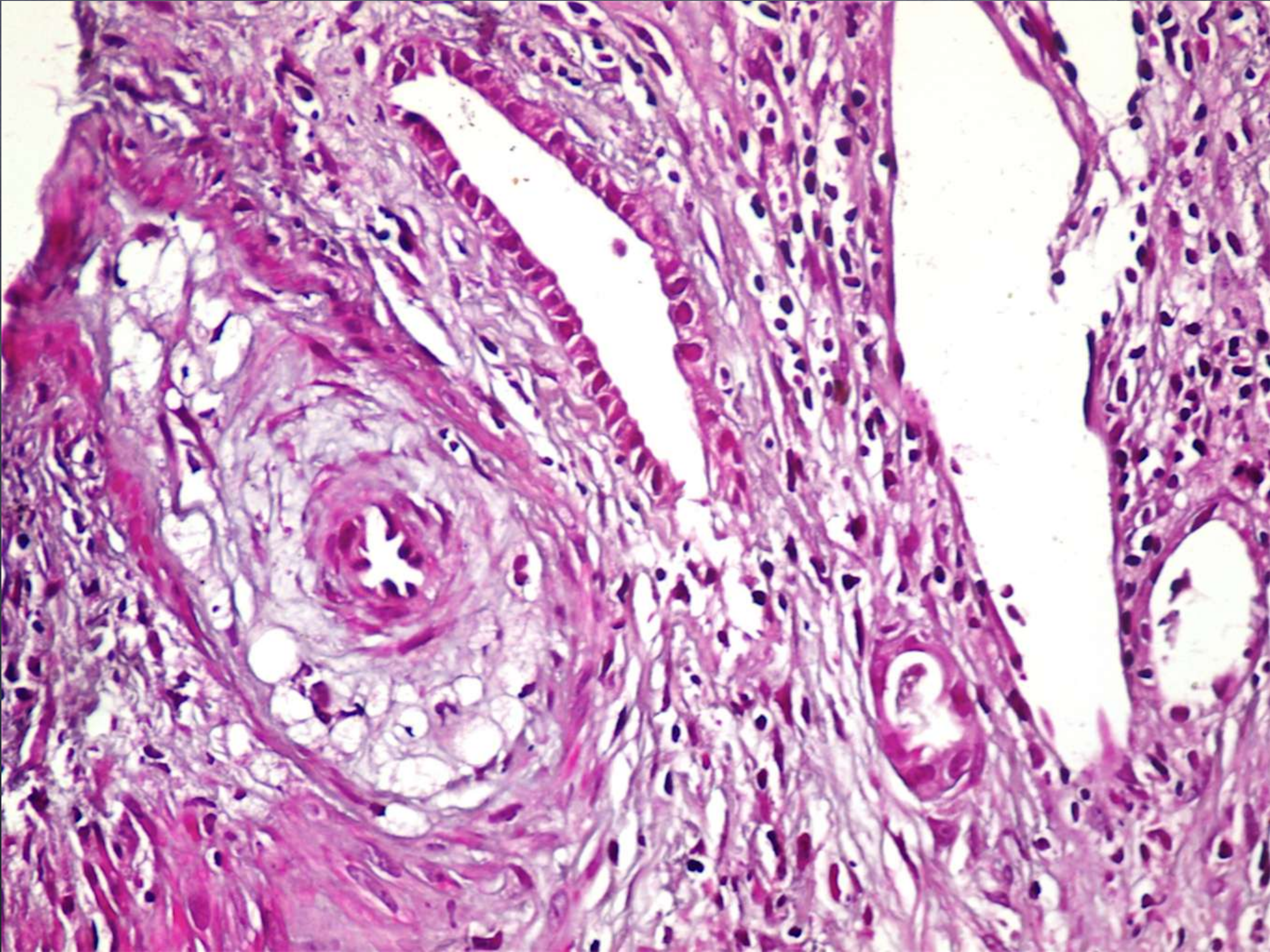


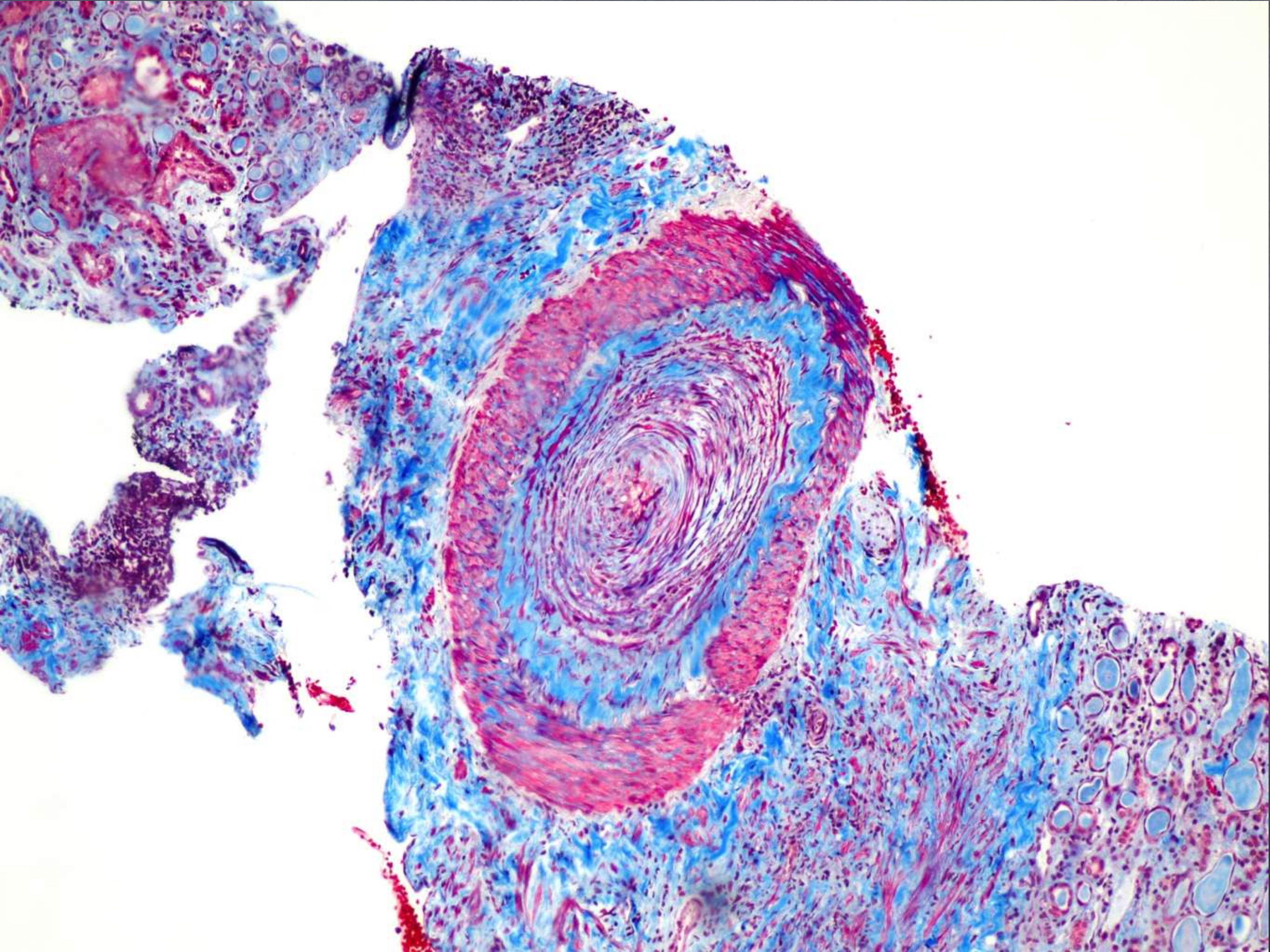


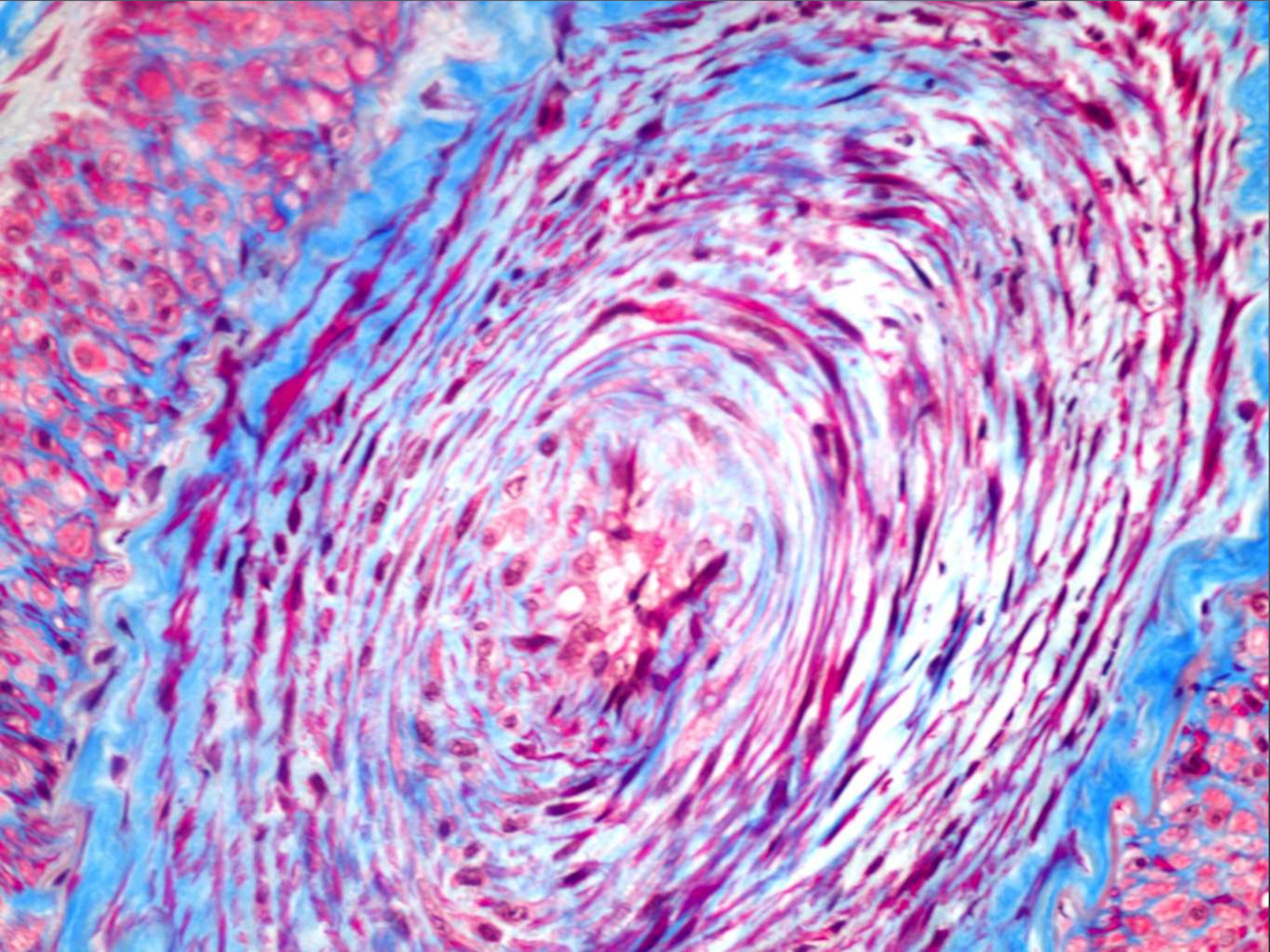






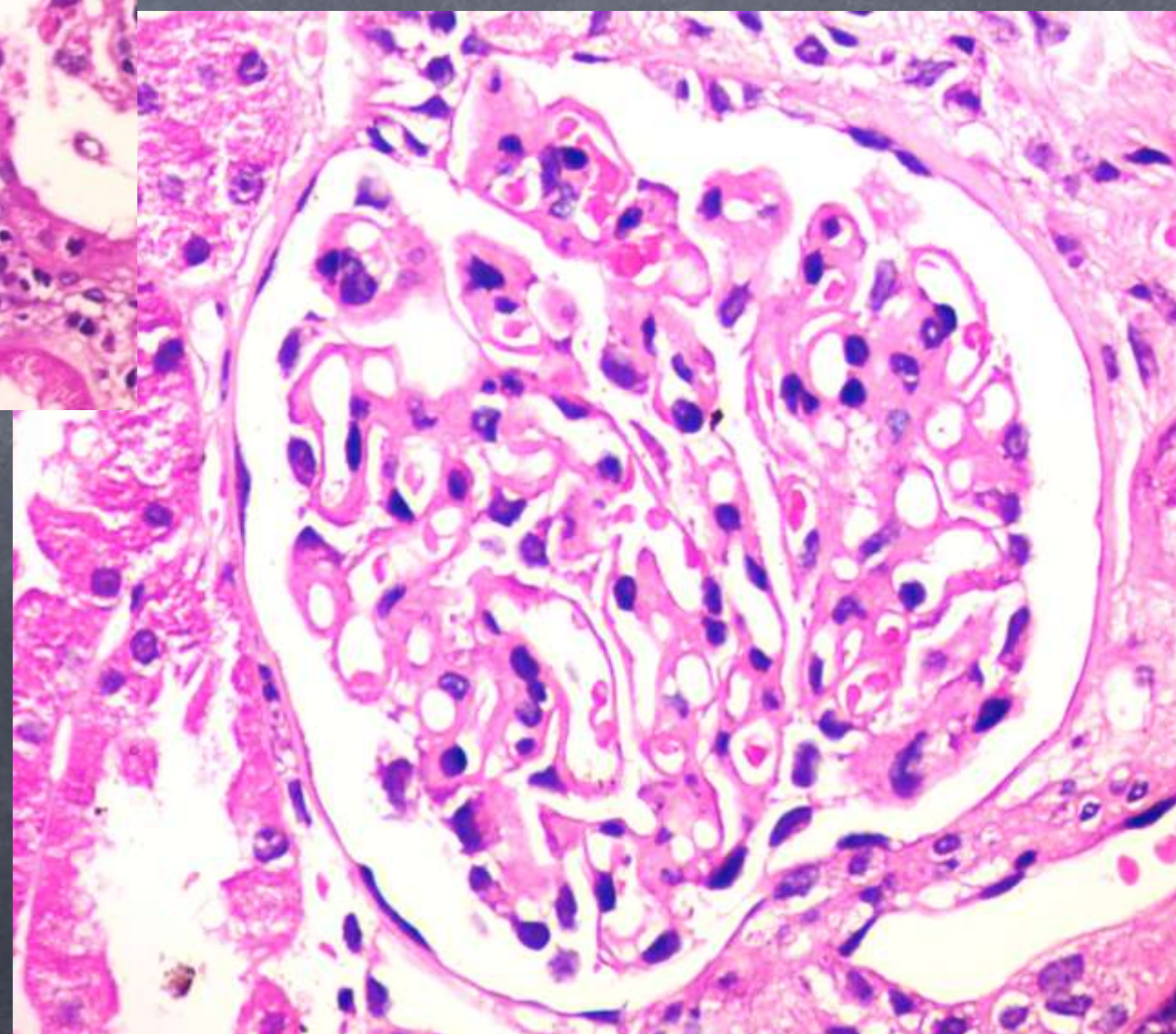
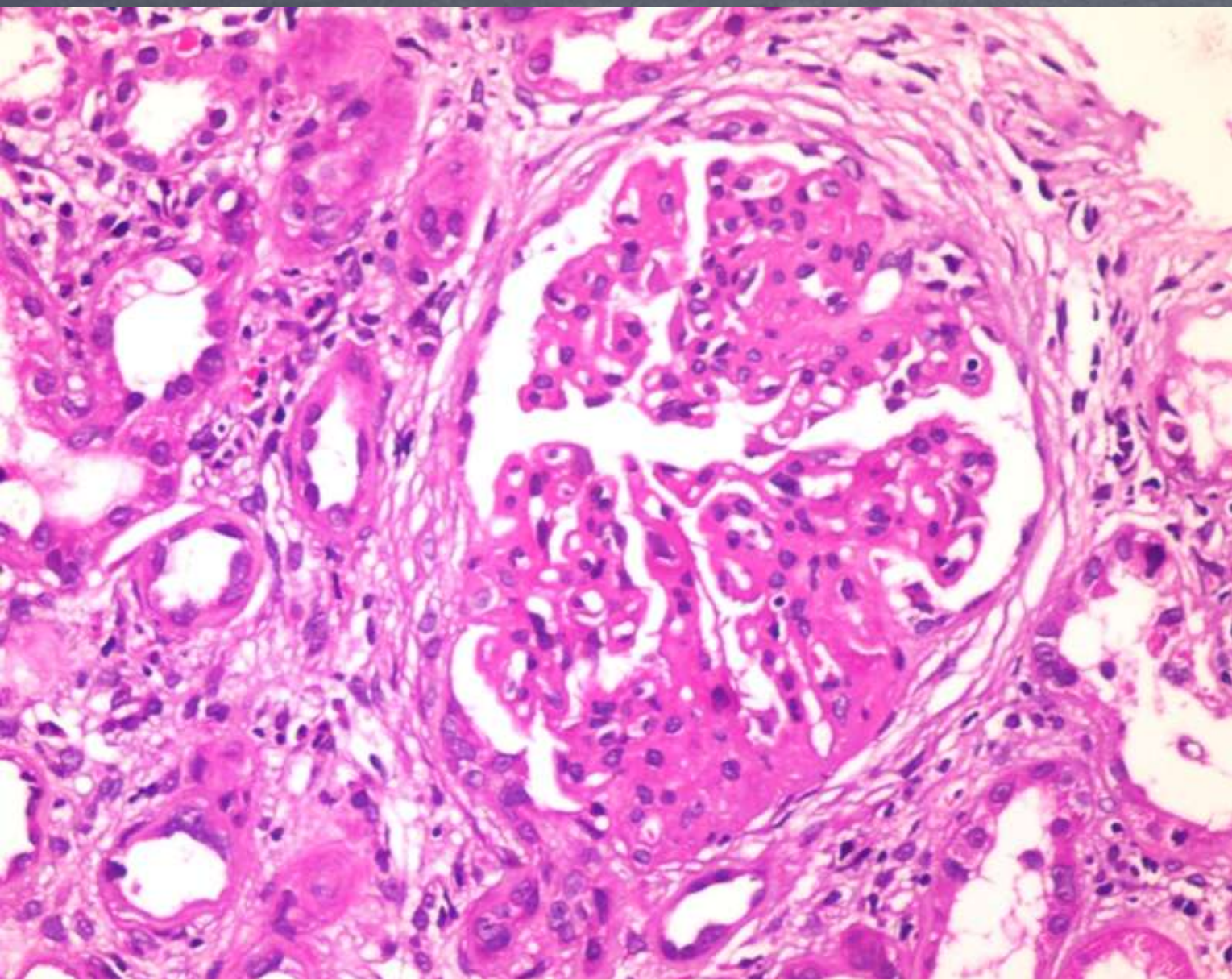


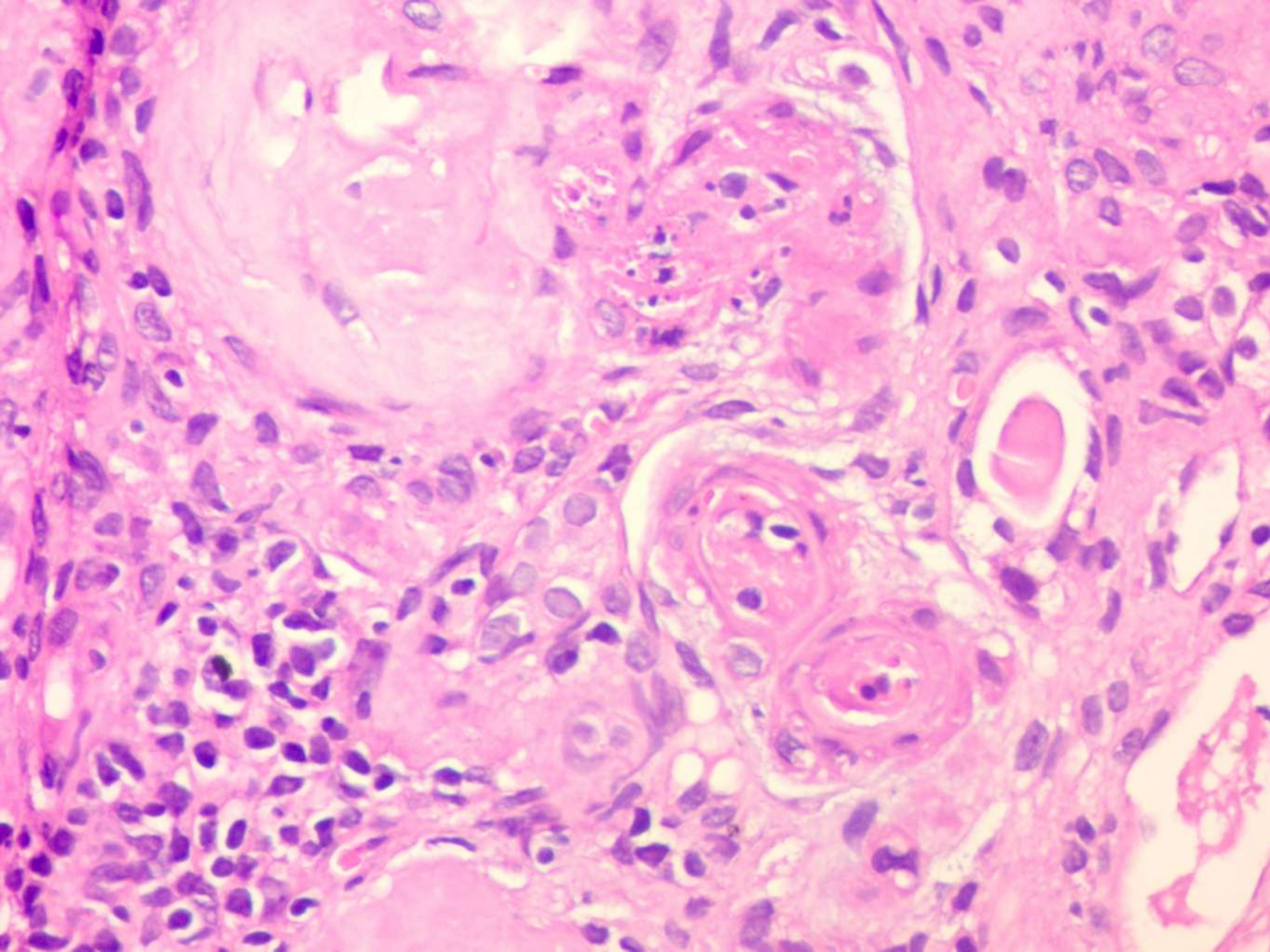


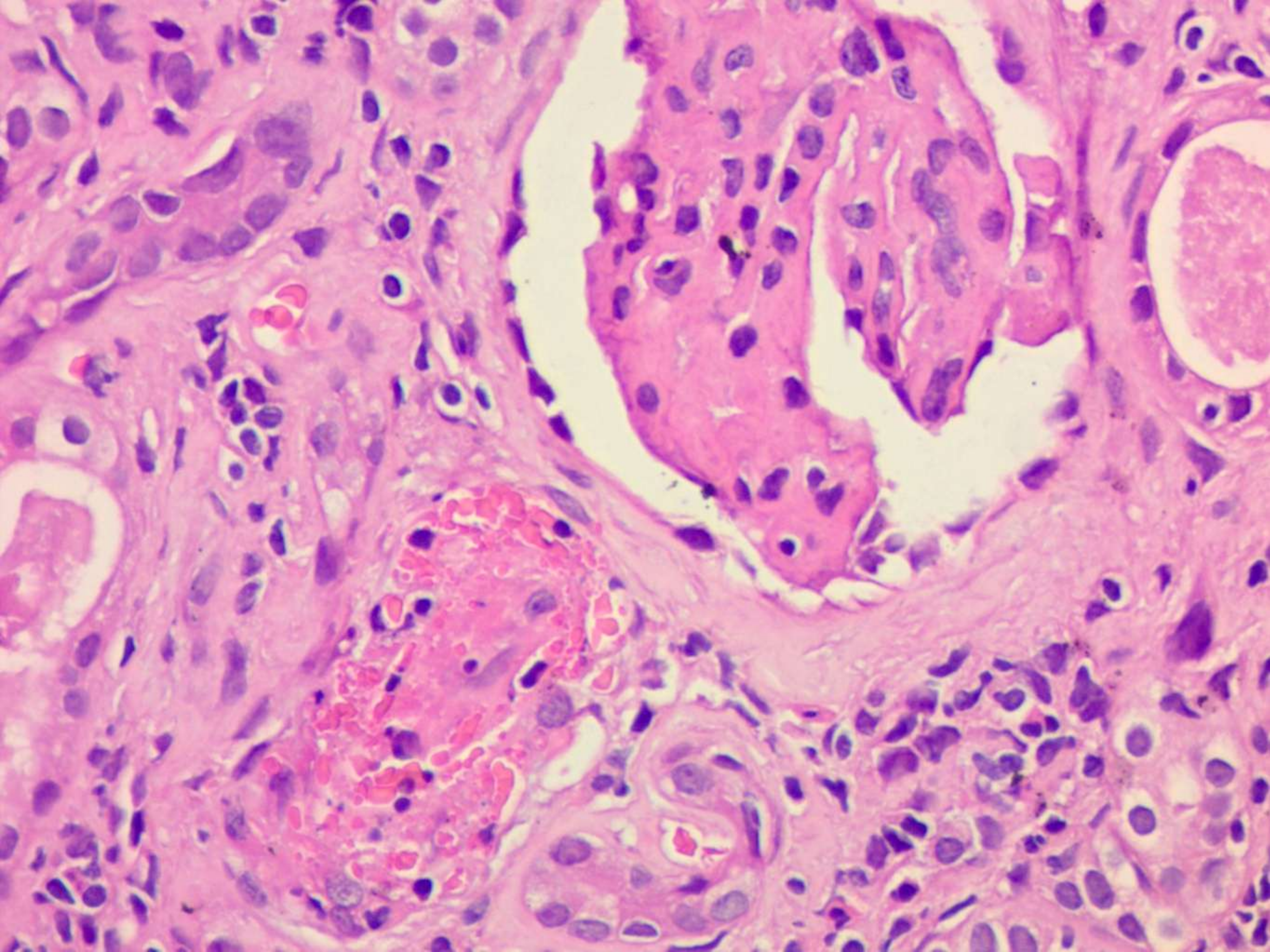


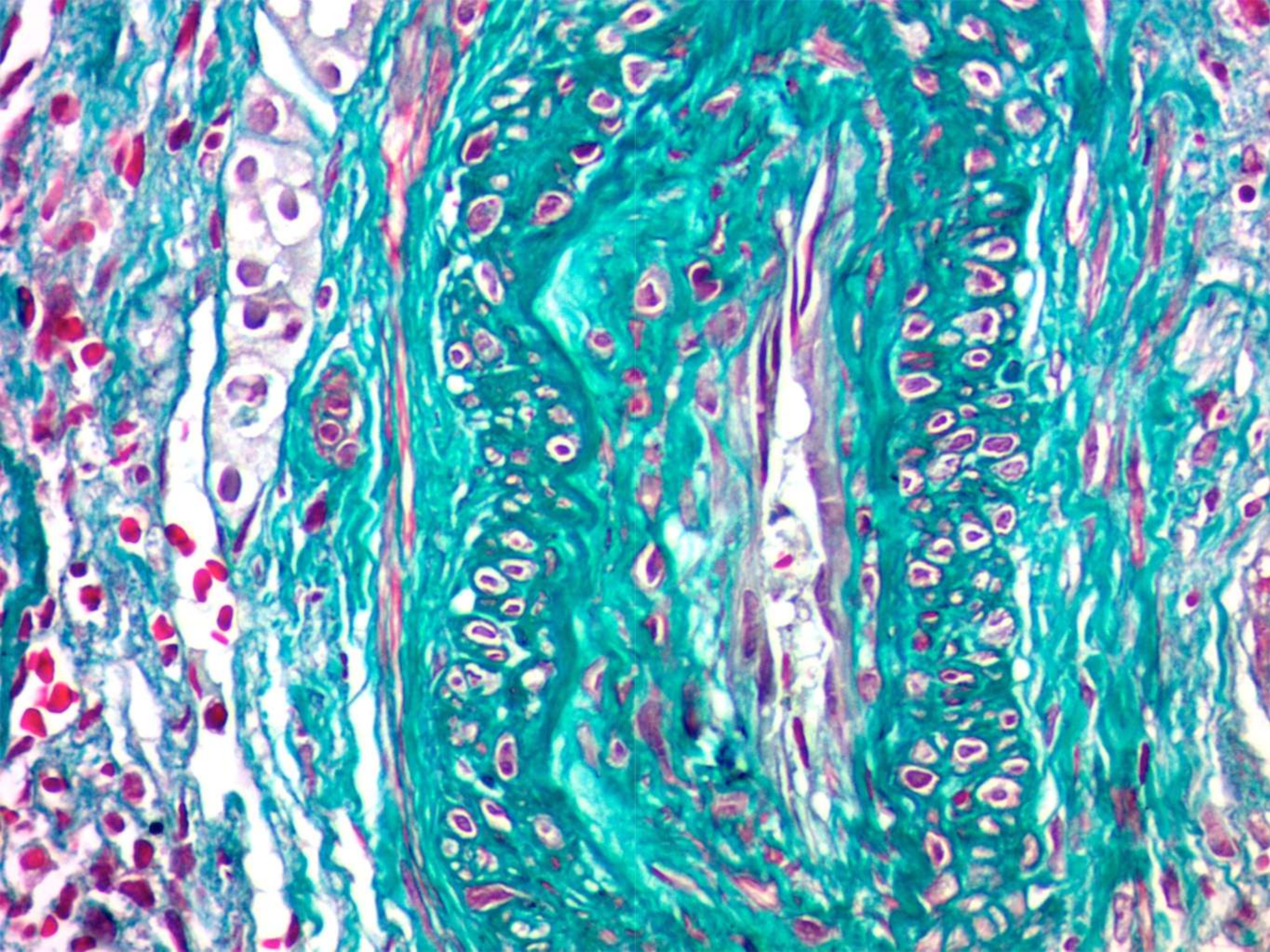
Case 2

- ❑ Female 43 years old, presenting with sudden increase of serum creatinine from 1.9 to 5.5 then 8 mg%
- ❑ Past history of nephrotic syndrome 4 years ago and was diagnosed as Membranous Nephropathy by biopsy (outside)
- ❑ She is known hypertensive more than 3 years, Not Diabetic
- ❑ 24 hours proteinuria:3.9 g/d
- ❑ Immunology and virology negative
- ❑ Renal biopsy



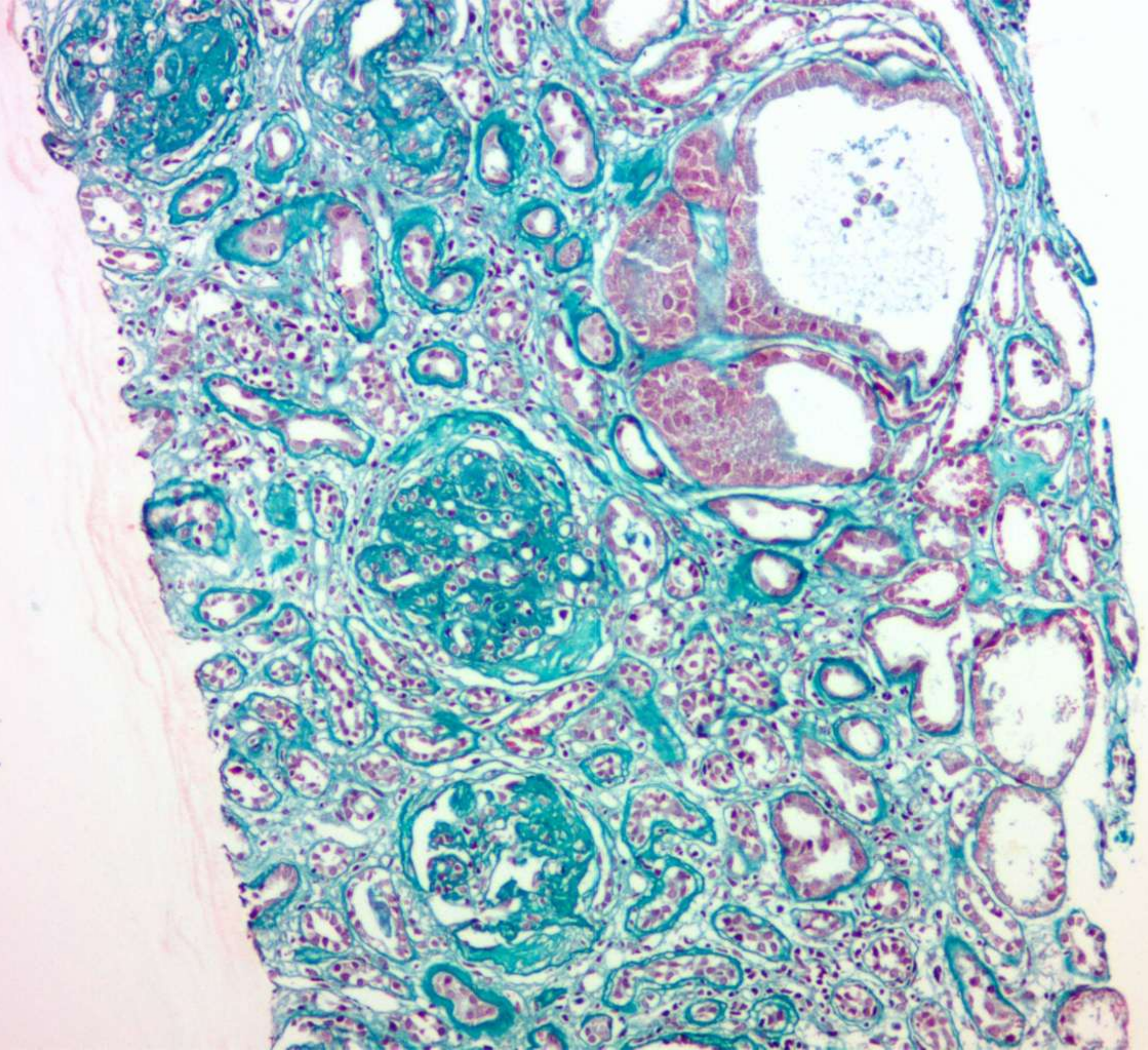


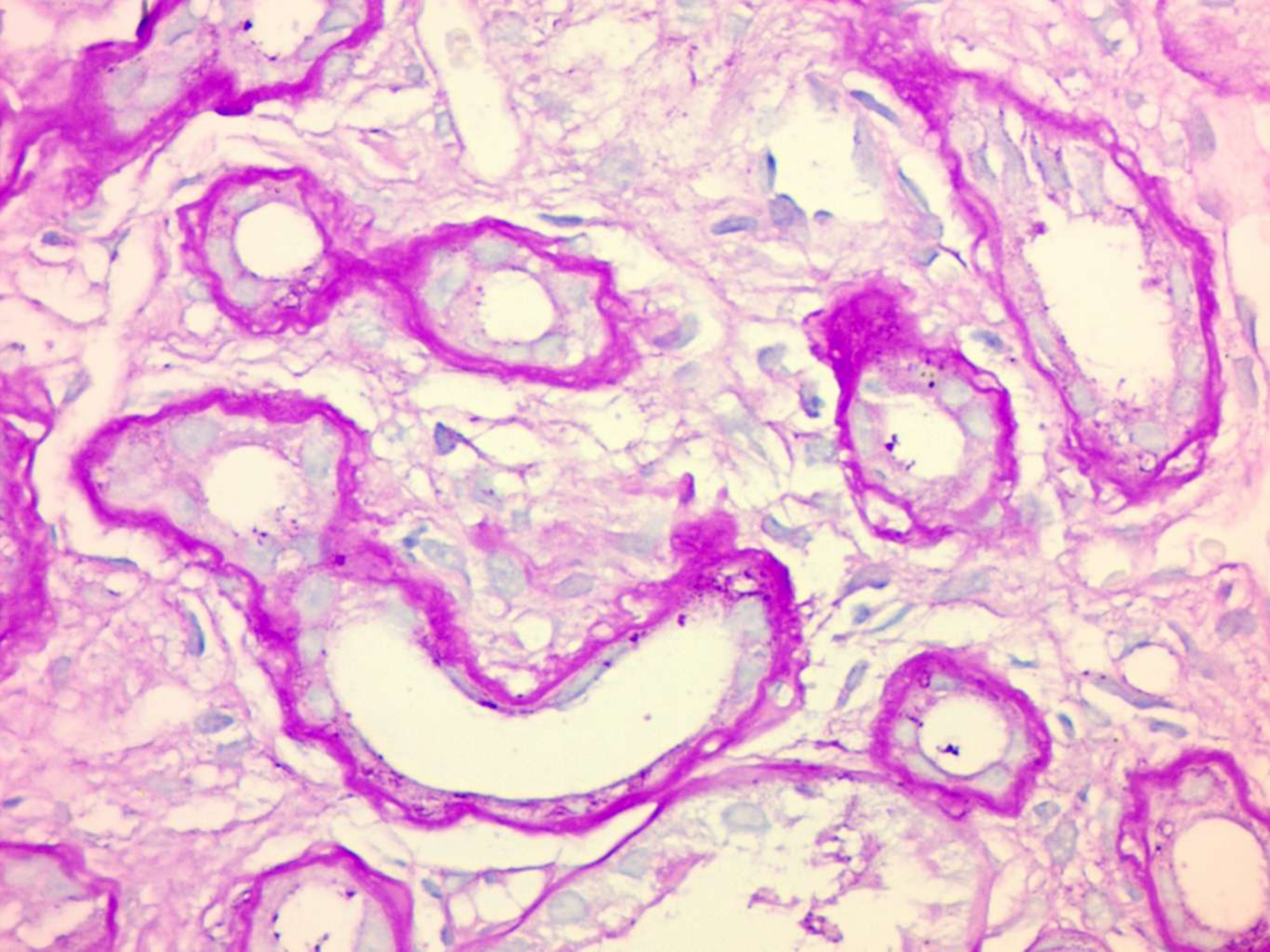




Accelerated Hypertension and Preexisting renal disease

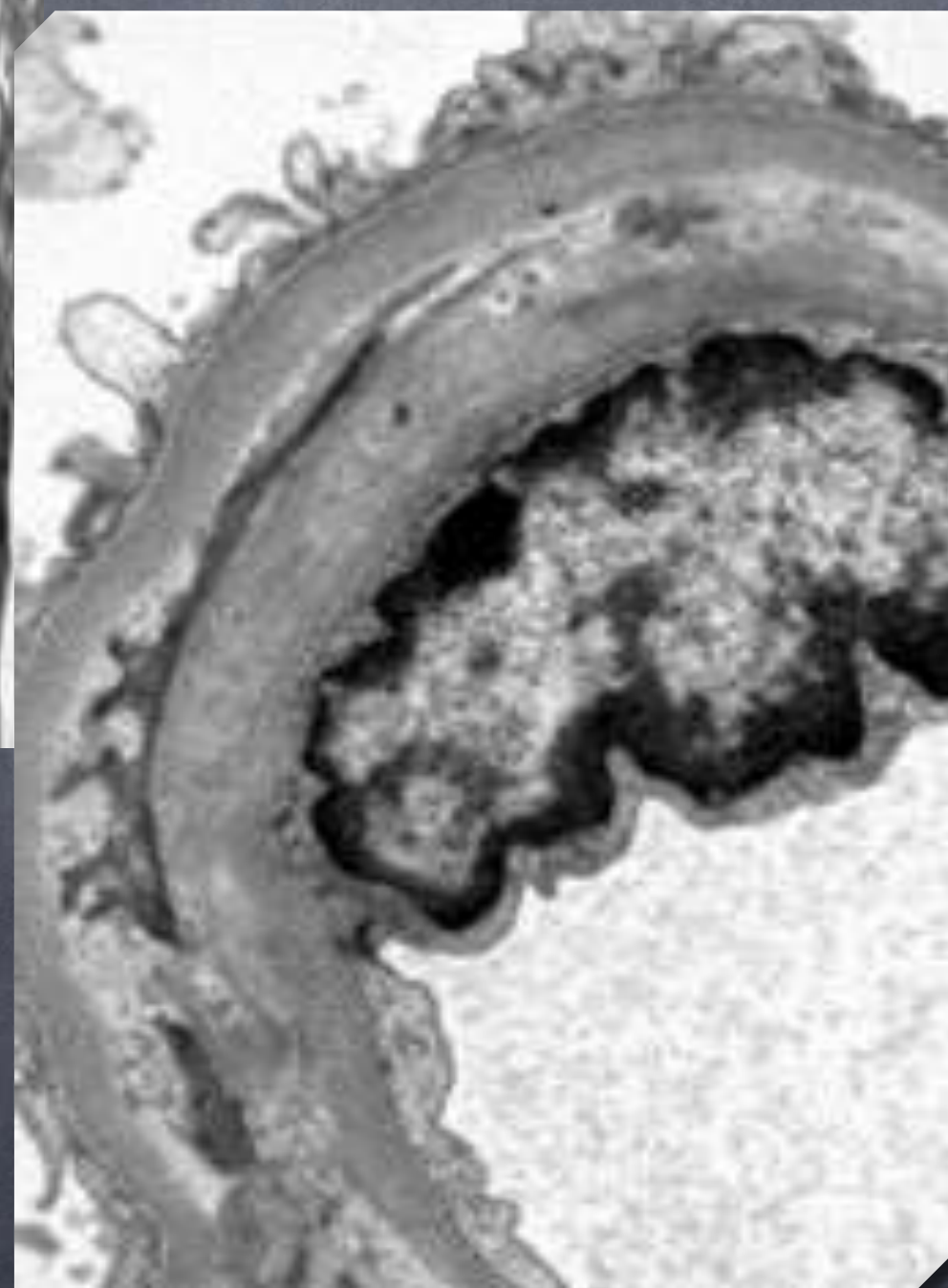
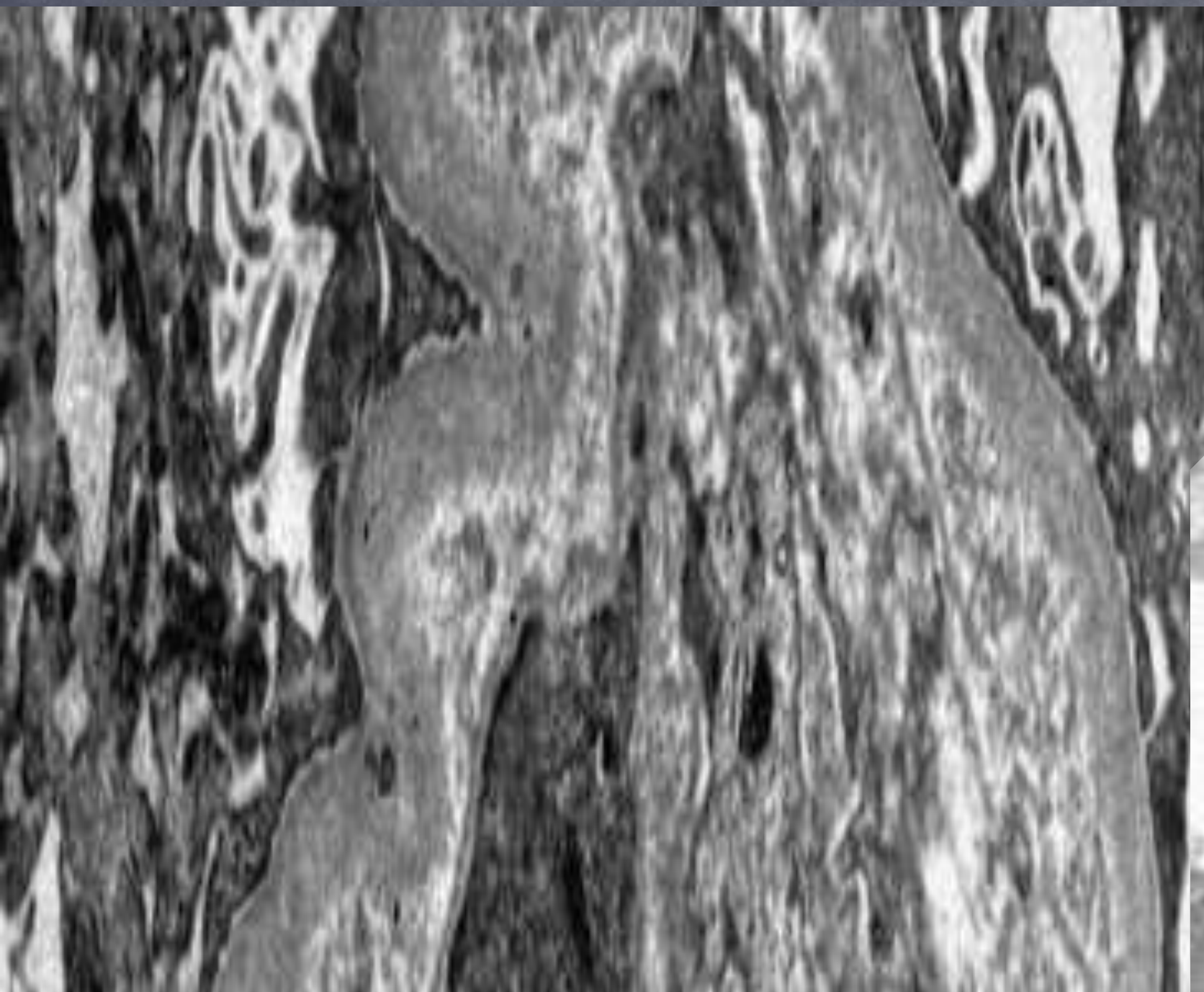
- The question of the primacy of malignant levels of hypertension in the pathogenesis of these microangiopathic lesions is still controversial
- Changes of “malignant” nephrosclerosis are superimposed on the kidneys with primary glomerular disease
- In these patients, usually severe renal parenchymal disease is present, and the history is consistent with preexistence of the glomerular disease before the onset of the malignant levels of hypertension
- Thus, maybe they can be considered as cases of secondary malignant hypertension





IF

- Arterioles with hyaline accumulation have been shown to contain IgM and C3 most commonly, but IgG, IgA, IgE, and fibrinogen have also been recorded
- C3 may also be observed without accompanying immunoglobulins
- The most common reactant seen in immunofluorescence studies of malignant hypertension is fibrinogen
- Fibrinogen is also seen in glomerular capillary loops and in larger vessels without obvious fibrinoid necrosis



Take away Message

- ❑ Hypertensive nephropathy is still one of the major causes of ESRD
- ❑ Accelerated phase hypertension can develop in patients with benign nephrosclerosis and in pre-existing renal disease
- ❑ Accelerated phase hypertension presents as “Thrombotic Microangiopathy”
- ❑ Thrombotic Microangiopathy is not HUS

THANK YOU FOR YOUR ATTENTION

? ?